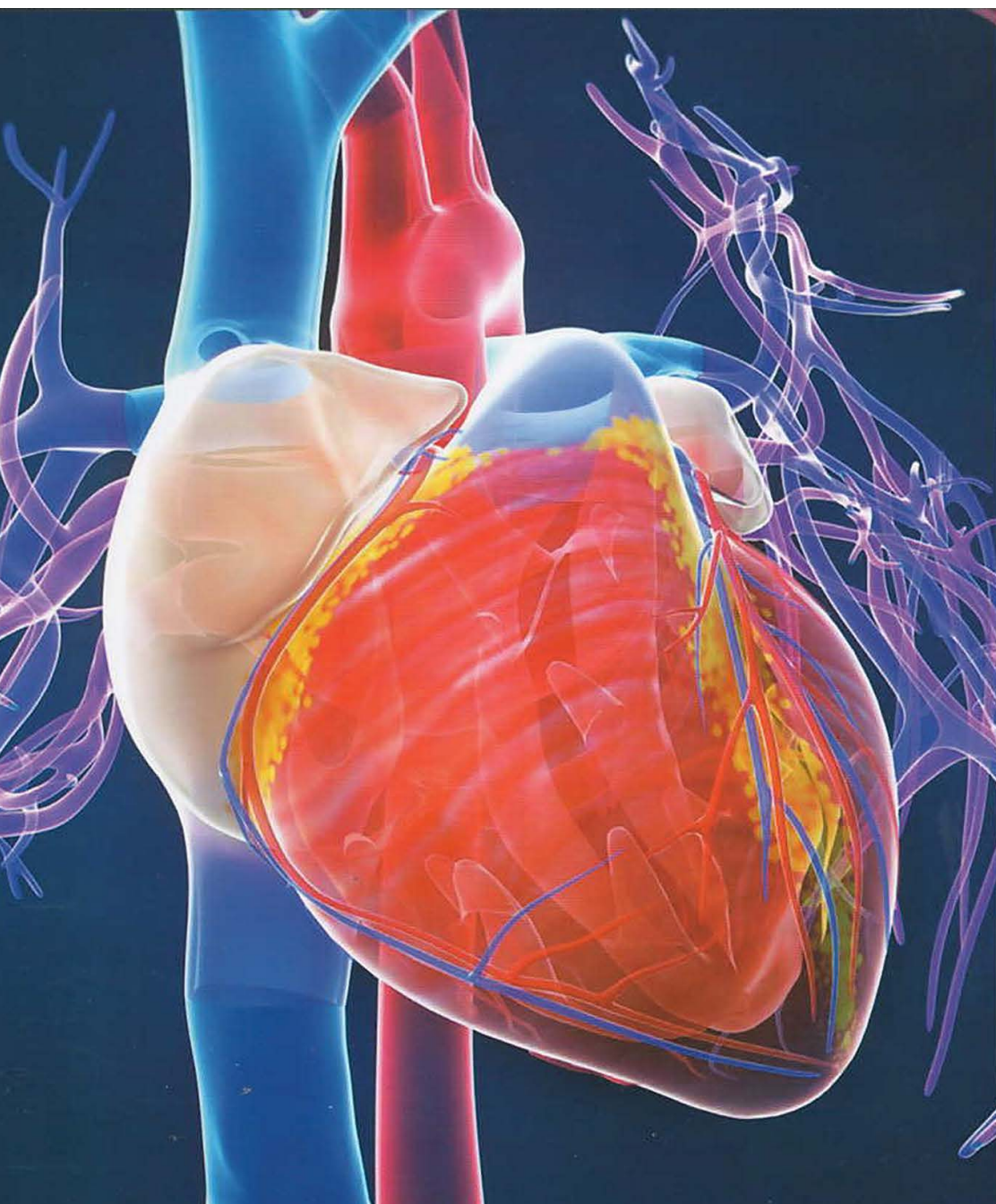


InCapsule | Series

SMARTER NOT HARDER

CARDIOLOGY



NOVAFY

INTERNAL MEDICINE



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In Capsule Series

Internal medicine

*“Working Smarter,
not Harder!”*

Cardiology

Dr. Ahmed Mowafy

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

لَا يُكَلِّفُ اللَّهُ نَفْسًا إِلَّا وُسْعَهَا لَهَا مَا كَسَبَتْ وَعَلَيْهَا مَا اكْتَسَبَتْ رَبَّنَا لَا تُؤَاخِذْنَا إِنْ
نَسِينَا أَوْ أَخْطَأْنَا رَبَّنَا وَلَا تَحْمِلْ عَلَيْنَا إَصْرًا كَمَا حَمَلْتَهُ عَلَى الَّذِينَ مِنْ قَبْلِنَا رَبَّنَا وَلَا
تَحْمِلْنَا مَا لَا طَاقَةَ لَنَا بِهِ وَاعْفُ عَنَّا وَاعْفِرْ لَنَا وَارْحَمْنَا أَنْتَ مَوْلَانَا فَانصُرْنَا عَلَى
الْقَوْمِ الْكَافِرِينَ

صدق الله العظيم

سورة البقرة (آية 286)

Preface

- *First and foremost, thanks are due to **ALLAH**, to whom I relate any success in achieving any work in my life.*
- *Great thanks to those who helped me, and greater thanks to those who try to break my wings as they make me more able to fly.*
- *My thanks are extended to all my medical students whom I produce this series "**In Capsule Series**" to obtain a higher degree in internal medicine by a simple effort.*
- *Finally, I wish to thank all members of My Family, my colleagues , my Friends and even all my patients for their continuous help, encouragement and support.*

Ahmed Mowafy

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1

General Scheme

Definition : *especially as regard to :*

- Heart failure.
- Ischemic heart disease.
- Hypertension.

Etiology: *enumeration*

Clinical picture : *7 items*

i. **Manifestations of LCOP :** *7 items*

Caused by deficient blood supply to :

- 1- **CNS** : Dizziness , headache , syncope .
- 2- **CVS** : Ischemic heart disease .
- 3- **Kidney** : Oliguria .
- 4- **Skin** : Cold , peripheral cyanosis .
- 5- **Skeletal muscle** : Fatigue , intermittent claudication.
- 6- **Blood pressure** : Low systolic blood pressure .
- 7- **Pulse** : weak.



ii. **Manifestations of Lung congestion :** (in Left sided diseases) *7 items*

- 1- **Dyspnea.**
- 2- Exertional cough .
- 3- Recurrent chest infections.
- 4- Hemoptysis.
- 5- Pleural effusion.
- 6- Pulmonary edema .
- 7- Bilateral basal crepitations.

iii. **Manifestations of systemic congestion :** (In **right** sided diseases) 7 items

- 1- Insomnia.
- 2- Sweating on slight activity.
- 3- Congested neck vein.
- 4- Edema lower limb , later on ascites.
- 5- Liver : enlarged ,soft, tender.
- 6- GIT : dyspepsia.
- 7- Pleural effusion.

iv. **chest pain :** Comment on the following points : 7 items

1. Site ?
2. Character ?
3. Radiation ?
4. Duration ?
5. Precipitating factors ?
6. Relieving factors ?
7. Association ?

v. **Palpitation** (sensation of own heart beats)

vi. **pressure manifestations :**

Resulting from **compression** e.g. : Left atrial enlargement, Pericardial effusion , Aortic aneurysm.

pressure symptoms are :

- Compression on **trachea** ⇒ cough & dyspnea.
- Compression on **esophagus** ⇒ dysphagia.
- Compression on **left recurrent laryngeal nerve** ⇒ hoarseness of voice.

vii. **Manifestations of embolization :** Sudden blindness, Hemiplegia

Investigations :

6 items

1- Xray :

- Chamber enlargement.
- Pulmonary congestion in left sided diseases.
- Pleural effusion.

**2- ECG :**

- Chamber enlargement.
- Detect the cause.

*“Working Smarter,
not Harder!”*

3- Echo :

- Chamber enlargement.
- Detect the cause.
- Paradoxical movement of the myocardium.

4- Catheterization :

- Chamber enlargement.
- Detect the cause.

5- Other imaging (radio isotope, CT, MRI) : may be needed.

6- Laboratory : CBC, Lipid profile, blood glucose, liver & renal function tests

But : add

- In **myocardial infarction** ⇒ *Cardiac enzymes.*
- In **infective endocarditis** ⇒ *Blood culture.*
- In **pulmonary embolism** ⇒ *Pulmonary angiography ,spiral CT , D dimer.*
- In **Cardiomyopathy** ⇒ *Biopsy.*

Treatment :

1- Treatment of the cause.

2- Treatment of precipitating factors : e.g. hyperlipidemia :

- Statins : 20 - 80 mg/d SE: myositis.
- Fenofibrate : 300 mg/d
- Omega 3 (fish oil) .

3- Specific treatment : **unfortunately it differs according to the disease !!!!**

2

Heart Failure

Definition:

- It is a **clinical syndrome** in which the heart can't pump an adequate cardiac output to meet the metabolic needs of the body.

Classification:

1- Left sided	Right sided	Both (<i>congestive HF</i>)
2- Systolic	Diastolic	Both
3- Acute	Chronic	Acute on top of chronic
4- Low COP	High COP	
5- Latent	Overt	
6- New York Heart Association (NYHA) Functional Classification.		
7- ACC/AHA stages of heart failure		
8- Framingham criteria for diagnosis of congestive heart failure		

Etiology:

I- Left – sided heart failure :

A) Left atrial failure: MS , Myxoma .

B) Left ventricular failure: 3 x 3

1- Muscle disease :

- **Myocardial infarction**
- **Myocarditis**
- **CardioMyopathy**

The most common 4 causes :

- IHD
- Systemic hypertension
- Valvular heart diseases
- Cardiomyopathy

2- Volume (diastolic) overload: (↑preload)

- Hyperdynamic circulation e.g. anemia, hyperthyroidism, Beriberi.
- Valvular disease: MR, AR
- Congenital disease: VSD, PDA

3- Pressure (systolic) overload: (↑ afterload)

- Systemic hypertension.
- AS.
- Coarctation of aorta.

II- Right sided heart failure:

A) Right atrial failure: TS , Myxoma

B) Right ventricular failure: 3 × 3

1- Muscle disease: The same as left .

2- Volume (diastolic) overload:

- Hyperdynamic circulation
- Valvular disease : TR, PR
- Congenital disease: VSD, ASD

3- Pressure (systolic) over load:

- Pulmonary hypertension.
- Pulmonary stenosis.
- Pulmonary embolism.

The most common causes of LSHF are :

- Ischemic heart disease is the most common cause of systolic HF.
- Systemic hypertension is the most common cause of diastolic HF.

The most common cause of RSHF is : LSHF

Diastolic heart failure : See P 22

In this type of HF, the decrease in COP is due to inadequate ventricular filling, not impaired systolic contraction. Most cases of diastolic failure are the result of hypertension induced left ventricular hypertrophy. *See below*

High cardiac output HF:

HF with hyperdynamic circulation e.g. Thyrotoxicosis, anemia.

Precipitating factors:

2I, 2P, 2A

- Infections: chest infections, infective endocarditis .
- Iatrogenic: Calcium channel blocker (- ve inotropic) .
- Cortisone (salt & water retention) .

NB: Calcium channel blockers like verapamil are effective in diastolic failure caused by idiopathic hypertrophic cardiomyopathy. MCO

- Physical & emotional stress. ➤ Pregnancy & delivery.
- Anemia. ➤ Arrhythmias (tachy & brady arrhythmias) .

Cardiac reserve (Compensatory mechanism) :**Aim:**

- ✓ To maintain normal COP.
- ✓ They are beneficial **within limit**. If they exceed these limits, they will aggravate HF

1- Reflex tachycardia: due to sympathetic ↑↑

2- Ventricular Dilatation: ↑ Volume load → increased length of cardiac muscle fibers → ↑ contraction within limit (*starling's law*)

3- Ventricular Hypertrophy: ↑ Pressure load → increased thickness of cardiac muscle fibers → ↑ contraction within limit. (*bigger is not better*)

4- Redistribution of blood flow:

From less vital organs (skin) to more vital organs (brain & heart)

5- Activation of renin - Angiotensin - Aldosterone System:

Hypovolemia → ↑renin → ↑Angiotensin II → ↑Aldosterone
→ Na & water retention → Hypervolemia .

6- Release of natriuretic peptide: (ANP , BNP)

Stretch of cardiac muscle fibers → Release of natriuretic peptide

VD & increase urinary Na excretion

Clinical Picture:**I- Left sided heart failure :** Scheme + cardiac signs**1- Manifestations of LCOP :** (Forward failure) 7 items

- 1- CNS : Dizziness, headache, syncope .
- 2- CVS : Ischemic heart disease.
- 3- Kidney : Oliguria .
- 4- Skin : Cold, peripheral cyanosis .
- 5- Skeletal muscle : **fatigue** , intermittent claudication .
- 6- Blood pressure: low systolic blood pressure.
- 7- Pulse : Weak.

2- Manifestations of pulmonary congestion: (Backward failure) 7 items

- 1- **Dyspnea**: Exertional , orthopnea, **paroxysmal nocturnal dyspnea** or dyspnea at rest. (mechanisms : see dyspnea)
- 2- Exertional Cough.
- 3- Recurrent chest infections.
- 4- Hemoptysis.
- 5- Pleural effusion.
- 6- Pulmonary edema .
- 7- Bilateral basal crepitations.

3- Features of the cause: Ischemic heart diseases , Systemic hypertension.**4- Cardiac signs:**

- a- Left ventricular enlargement :
- b- Tachycardia.
- c- Pulsus alternans: alternating strong & weak beats (In advanced stage)
- d- Gallop on the apex: due to flabby ventricle.

- 1- Localized apex.
- 2- Shifted apex out & down.
- 3- Systolic bulge.

ventricular gallop = S₃ + tachycardia

- e- Murmur of functional MR: pansystolic murmur due to LV dilatation .

II **Right sided heart failure:**

1-Manifestations of LCOP : *see before*

2-Manifestations of Systemic congestion :

- 1- Insomnia.
- 2- Sweating on slight activity (diaphoresis) : due to sympathetic activation.
- 3- Congested neck vein.
- 4- Edema lower limb, later on ascites.
- 5- Liver: enlarged, soft, tender.
- 6- GIT: dyspepsia, malabsorption → may lead to **cardiac cachexia**.
- 7- Pleural effusion.

3-Features of the cause: e.g. LSHF , Pulmonary hypertension .

4-Cardiav Signs: (the same as left – pulsus alternans)

- 1- Right ventricular enlargement : 6
- 2- Tachycardia
- 3- Gallop (over tricuspid area).
- 4- Murmure of functional TR .

N.B. LSHF → Lung Congestion.
RSHF → Systemic Congestion.

Right ventricular enlargement : 6

- 1- Diffuse apex. 2- Shifted apex outward 3- Systolic retraction.
- 4- Wide bare area. 5- Left parasternal pulsation. 6- Epigastric pulsation.

Clinical classification of HF :

New York heart association functional classification :

I	No limitation of physical activity (no symptoms with ordinary activities.
II	Slight limitation (symptoms with ordinary activities)
III	Marked limitation (symptoms with less than ordinary activities)
IV	Severe limitation (symptoms of heart failure at rest)

Framingham criteria for diagnosis of congestive heart failure :

Diagnosis of CHF requires the presence of at least 2 major criteria or 1 major criterion in conjunction with 2 minor criteria.

Major criteria :

- Paroxysmal nocturnal dyspnea
- Neck vein distention
- Rales
- Radiographic cardiomegaly.
- Acute pulmonary edema
- S₃ gallop.
- Increased central venous pressure (>16 cm H₂O at right atrium)
- Hepatojugular reflux
- Weight loss >4.5 kg in 5 days in response to treatment.

Minor criteria :

- Bilateral ankle edema
- Nocturnal cough
- Dyspnea on ordinary exertion
- Hepatomegaly
- Pleural effusion
- Decrease in vital capacity by one third from maximum recorded
- Tachycardia (heart rate >120 beats/min.)

ACC/AHA stages of heart failure :

(The American College of Cardiology/American Heart Association classification of heart failure)

Stage 1	High risk for heart failure without structural heart changes (those with diabetes, HTN, CAD). No symptoms of heart failure.
Stage 2	Patients with structural heart disease (i.e. reduced ejection fraction, left ventricular hypertrophy, chamber enlargement) who have not yet developed symptoms of heart failure.
Stage 3	Patients who have developed clinical manifestations of heart failure.
Stage 4	Patients with refractory heart failure requiring advanced intervention (i.e. biventricular pacemakers, left ventricular assist device, transplantation).

Differential Diagnosis :

LSHF	RSHF
-Causes of dyspnea & orthopnea.	- Pericardial effusion. - COPD. - Obesity. - Liver cirrhosis.

Complications of heart failure :

- Renal failure.
- Liver cell failure : due to associated hepatitis C & cardiac cirrhosis.
- DVT & Pulmonary embolism.
- Valvular heart disease e.g. MR
- Cardiac cachexia especially with RSHF.
- Acute pulmonary edema.
- Stroke, syncope.
- Arrhythmias : atrial & ventricular.

Investigations:**1- X ray :**

- Chamber enlargement.
- Pulmonary congestion in LSHF.

In one

Investigations of HF :
Scheme + **Echo** (EF) + BNP

2-ECG : *Usually nondiagnostic*

- Detect the cause e.g. MI, arrhythmias
- Chamber enlargement.

3-Echocardiography: (key investigation)

- Chamber enlargement & detect the cause.
- Paradoxical movement of the myocardium.
- Measurement of **Ejection fraction** (EF) & Shortening fraction (SF)

$$\text{Ejection fraction} = \frac{\text{Stroke volume}}{\text{End diastolic volume}} \quad (n = 50\%)$$

EF < 45% → systolic HF

Shortening fraction : (SF)

- It measures the change in diameter between the relaxed state and constricted state of the left ventricle.
- It is calculated by the left ventricle end-diastolic and end-systolic diameters.

$$\text{Shortening fraction} = \frac{(\text{LVEDD} - \text{LVESD})}{\text{LVEDD}} \times 100 \quad (N : 30-42\%)$$

- LVEDD (Left ventricular end-diastolic dimension) : 36 - 56 mm

- LVESD (Left ventricular end-systolic dimension) : 20 - 40 mm

**4-Cardiac catheterization:**

- Chamber enlargement.
- Detect the cause.

5- Other imaging (Nuclear cardiography, CT , MRI) : may be needed.

6- Laboratory :

- CBC : to detect anemia.
- Liver function tests : may be impaired due to liver congestion.
- Renal function tests : may be impaired due to LCOP.
- Cardiac enzymes : in acute heart failure to diagnose MI.
- Electrolytes : may be impaired due to drugs.

7 - BNP (B Natriuretic Peptide, Ventricular Natriuretic Peptide) : > 500 ng/L

It is a good negative predictive value (if normal, HF is unlikely).

Treatment :

- A. Treatment of the underlying cause.
- B. Treatment of precipitation factors e.g. anemia

C. General treatment :

1-Rest:

- Until signs of HF disappear.
- Semisitting rather than lying down to decrease the venous return.
- complications of prolonged bed rest:
 - Psychosis.
 - Bed sores.
 - Pulmonary embolism.
 - DVT.
 - Constipation.
 - Retention of urine.

2-Diet :

- Salt restriction is essential.
- Fluid restriction: in severe cases.
- Low calories.
- Small frequent meals, ↑ K.

3-Sedation : as diazepam.

4- Life style modification : e.g. mild exercise may be of value.

D. Specific treatment :

1. **Medical.** " See below "

2. **Interventional.**

➔ **Cardiac resynchronization therapy (RCT)** : biventricular pacemaker to improve mechanical cardiac function.

➔ **Cardiac revascularization** : if reversible ischemia is detected.

3. **Surgical :**

➔ For the cause : e.g. CABG (*coronary artery bypass graft*), valve replacement.

➔ Cardiac assist devices.

➔ Cardiac transplantation.

Medical treatment :**I. Decrease preload :**

✎ Diuretics.

✎ Venodilators e.g. nitrate.

II. Decrease afterload :

✎ Arterial vasodilators e.g. hydralazine, Na nitroprusside.

III. Increase myocardial contraction : (Inotropes)

✎ Digitalis.

✎ β agonist e.g. Dopamine.

✎ Milrinone : phosphodiesterase inhibitor.

IV. Neuro-hormonal treatment :

✎ RAAS (renin-angiotensin-aldosterone system) inhibitors :

- Aldosterone antagonist :

- Spironolactone : potassium-sparing diuretic.

- Eplerenone : more specific, more expensive. It is effective in treatment of HF after myocardial infarction.

- ACE inhibitors & ARBs (Angiotensin receptor blockers).

✎ β blockers.

V. Others :

- ✎ Aminophylline.
- ✎ Anticoagulants : especially in patients with AF, or with previous history of thromboembolism.
- ✎ Oxygen therapy.

Diuretics :

➤ **Aim:** *Just symptomatic relief. No mortality benefit.*

- a. They increase salt & water excretion → ↓ blood Volume So, decrease preload .
- b. ↓ Edema & visceral congestion.

➤ **Types:**

i- Loop diuretics:

- Act on Loop of henle (↓ reabsorption of Na, H₂O, K, Cl)
- e.g. - Furosemide (**Lasix**) : 40-160 mg/d (oral, IV, IM).
- Bumetanide (**Burinex**)

ii- Thiazides:

- Act on distal tubules ↓ reabsorption of Na, H₂O, K, Cl)
- e.g. - Hydrochlorothiazide: 25-100 mg/d, Chlorothalidone.

iii- Potassium sparing diuretics : Spironolactone

- Can be combined with lasix or thiazide to avoid hypokalemia.

➤ **Side effects of lasix & thiazides:**

4 hypo:

- hypokalemia
- hypovolemia
- hyponatremia
- hypochloremic alkalosis

-4 hyper (glucose)

- hyperglycemia
- hyperlipidemia
- hyperuricemia
- hypercalcemia (Thiazide only)

Loop diuretics (Lasix) Lose Ca, whereas thiazides save it.

🗑 **Lasix** → Ototoxicity & nephrotoxicity.

🗑 **Spirolactone** → Hyperkalemia & gynecomastia.

- In HF: lasix is more better than thiazide.
- Better given in the morning. It's better to combine diuretics with ACEIs.

Vasodilators: They are *classified* into:

Arteriolar	Venous	Both
- Reduce afterload	Reduce preload	Reduce both.
- Hydralazine	Nitrates	- ACEIs.
- Diazoxide		- Na nitroprusside.

Pharmacological details: *see systemic hypertension*

- ➡ ACE inhibitors are now the **cornerstone** treatment of HF.
- ➡ They are effective in prevention, symptoms control and **improve mortality** & morbidity. **MCQ**

Inotropic agents :

- **Digitalis.**
- Dopamine, Dobutamine.
- **Milrinone** : phosphodiesterase inhibitors, used in emergency.

Digitalis

➤ **Action :**

- ↑↑ Contractility of the ventricles.
- ↑↑ Excitability.
- ↓↓ Conductivity.
- ↓↓ HR : *by direct action & vagal stimulation.*
- **On ECG** : sagging depression of ST segment.

➤ **Mechanism of action :** (↑ contractility)

Inhibition of Na - K ATPase (Na pump) → ↑ intracellular Na → ↑ intracellular Ca → increase muscle contraction by sliding of actin & myosin.

➤ **Indications :**

- Its use in chronic HF had been become very **limited** nowadays.
- Digitalis should be used as first line therapy in patients with heart failure & atrial fibrillation.
- *Digitalis is of no benefit & may lead to life threatening toxicity in cases of heart failure with the following underlying heart diseases : MCO*

- ☠ *Pericardial tamponade & constrictive pericarditis.*
- ☠ *Coronary heart disease.*
- ☠ *Isolated MS.*
- ☠ *Pulmonary disease & RVF*
- ☠ *Hypertrophic cardiomyopathy & those with diastolic dysfunction due to decreased ventricular compliance.*

➤ **Contraindications:**

○ Absolute contraindications :

- Digitalis toxicity.
- Ventricular tachycardia (VT).

○ Relative contraindications :

- Partial heart block.
- Peptic ulcer.
- Nodal rhythm.

➤ **Administration :**

- Digitalization : (to reach optimum therapeutic level)
2 tablets daily for 5 days (oral).
- Maintenance dose : (compensates for daily urinary excretion)
0.5 – 1 tablets daily (oral)

➤ Preparations :

- Digoxin (Lanoxin): excreted mainly by the kidney (tab=0.25mg , amp=0.5mg)
- Digitoxin : metabolized mainly in the liver (digitoxine → hepatic).
- Ouabain (IV) .

DIGITALIS TOXICITY:

○ Precipitating factors :

MCQ

- Renal failure.
- Old age.
- Hypokalemia, Hypomagnesemia.
- Hypercalcemia, Hypernatremia.
- Hypothyroidism & hypothermia.
- Alkalosis & Acidosis.
- Myocardial ischemia.
- Drugs : quinidine, verapamil, amiodarone, sympathomimetics...

Al-k-lossis ⇌ Hypokalemia ☺

Both acidosis and myocardial ischemia suppress the Na^+/K^+ ATPase pump

○ Clinical picture:

Non cardiac :

- GIT : Anorexia , nausea, vomiting (*1ST symptom*)
- Neurological: Psychosis , headache, paresthesia , confusion.
- Visual : yellow-green vision, Photophobia
- Gynecomastia.

Cardiac : (*most life threatening*)

- ↑ excitability → Arrhythmias.
- ↓ AVN conduction → Heart block.

*Almost any arrhythmia can be a manifestation of digitalis toxicity except
type 2 second degree heart block & sinus tachycardia.* **MCQ**

○ Treatment :

- Stop digitalis.
- Stop diuretics.
- Give K in a case of hypokalemia.
- Replace Mg.
- Digitalis antibodies (Digibind) :
 - Binds to intravascular digitalis
 - It is used when the patient is hemodynamically unstable, or with life threatening arrhythmias.
- Symptomatic treatment :
 - Anti-arrhythmic drugs e.g. phenytoin, lidocaine.
 - Atropine for heart block.
 - Manage hyperkalemia : as usual except don't use Ca gluconate.

○ To avoid toxicity :

- Decrease the dose.
- Drug holiday.
- Routine estimation of serum level of digitalis ($N=0.5-2\text{ng/ml}$).

β-blockers :

- Historically, β blockers were contraindicated in HF due to their -ve inotropic effect.
- Recently : β blockers are indicated in HF because they were found to :
 - Improve mortality & morbidity.
 - Prevent or partially reverse progressive left ventricular dilatation.
 - Prevent arrhythmia.
 - Decrease blood pressure.
 - Decrease renin.

➤ **Only**, 3 drugs are used in treatment of HF :

- Metoprolol (2nd generation β_1 blocker)
- Bisoprolol.
- **Carvedilol** (3rd generation β blocker).

➤ Start with low doses with gradual increase.

Entresto (sacubitril/valsartan) : recent drug

- Entresto is a combination of :
 - Sacubitril: Neprilysin inhibitor. Neprilysin is responsible for degradation of ANP & BNP.
 - Valsartan: Angiotensin II receptor blocker.
- It is indicated to reduce the risk of cardiovascular death and hospitalization for heart failure in patients with chronic heart failure (NYHA Class II-IV).
- The recommended starting dose of ENTRESTO is 100 mg twice-daily. Double the dose of ENTRESTO after 2 to 4 weeks to the target maintenance dose of 200 mg twice daily, as tolerated by the patient.
- **Contraindications :**
 - ☠ Patients with hypersensitivity to any component.
 - ☠ Patients with a history of angioedema related to previous ACE inhibitor or ARB therapy.
 - ☠ With concomitant use of ACE inhibitors. Do not administer within 36 hours of switching from or to an ACE inhibitor.
 - ☠ With concomitant use of aliskiren (renin antagonist).

ACUTE HEART FAILURE

(Acute Cardiogenic Pulmonary Edema)

Etiology : (sudden ↑ in pulmonary venous pressure)

- Acute left sided heart failure e.g. myocardial infarction.
- On top of chronic LSHF : MS with aggravating factor as AF.

Clinical picture :

- Severe dyspnea at rest & orthopnea.
- Sense of impending death.
- Sweating & irritability.
- Cyanosis.
- Crepitation .
- Cough with frothy pink sputum.
- Features of the Cause : MI.

Differential Diagnosis :

- Non Cardiogenic pulmonary edema (ARDS).
- DD of acute dyspnea : see later

Treatment :

- 1) Hospitalization in ICU : bed rest in sitting position
- 2) High dose oxygen $\Rightarrow$ correct hypoxia
- 3) **Morphine** (IV) $\longrightarrow$ Reduce anxiety.
 $\longrightarrow$ Reduce preload (venodilator).
- 4) **Furosemide** (IV) $\longrightarrow$ Decreases pulmonary congestion (venodilator)
 $\longrightarrow$ Diuresis.

5) Vasodilators (IV):

- Na nitroprusside → IV infusion (0.5 – 5 mg / kg/ min)
- Nitroglycerin → IV infusion (S/E : tolerance).

6) Inotropics :

- Dobutamine (β receptor agonist) : +ve inotropic & vasodilatation (inodilator)
- Milrinone (phosphodiesterase inhibitor) : inodilator.

 NB

Milrinone is preferred to dobutamine in patients receiving β blocker because its mechanism of action does not involve β receptors. Dobutamine also may precipitate ischemic heart disease.

7) Aminophylline : 250 – 500 mg / IV infusion very slowly .

8) Treatment of the cause & the precipitating factors .

9) Advanced management : in refractory conditions

- Mechanical ventilation.
- Mechanical assist devices : Intra-aortic balloon counter pulsation.

You know you're in love when you don't want to fall asleep because reality is finally better than your dreams.

Dr. Seuss

Diastolic heart failure

Etiology : *Any condition that leads to stiffening of the ventricle*

- 1- **Hypertension.**
- 2- Aortic stenosis.
- 3- Hypertrophic cardiomyopathy.
- 4- Old myocardial infarction (*scarred heart muscle*)
- 5- Old age (*age alone causes stiffening of the ventricles*)
- 6- DM (*stiffening occurs as a result of glycosylation of heart muscle*).

Diagnosis :

- The same symptoms & signs of systolic heart failure but the left ventricular ejection fraction (EF) is normal.
- A second approach is to use an elevated BNP level in the presence of normal EF .

Treatment :

- 1- Treatment of the cause e.g. systemic hypertension , Ischemic heart diseases.
- 2- Specific treatment : The same as systolic heart failure but without digitalis.

- ✓ Digitalis and other inotropic agents have no established place in these patients with relatively normal ejection fraction.
- ✓ ACE inhibitors : cause regression of left ventricular hypertrophy, decrease blood pressure, and prevent cardiac remodeling.
- ✓ Beta blockers : decrease heart rate, increase diastolic filling time, decrease oxygen consumption, lower blood pressure, and cause regression of left ventricular hypertrophy.
- ✓ Ca channel blockers e.g. verapamil : effective only in diastolic failure caused by idiopathic hypertrophic cardiomyopathy. MCQ

Refractory (*Intractable*) Heart Failure

Patients don't improve or experience rapid recurrence of symptoms in spite of optimal medical therapy.

ETIOLOGY :

- **Diagnostic error** : the case may be pericardial effusion rather heart failure.
- **Improper management** :
 - Inadequate salt restriction.
 - Discontinuation of treatment.
- **Presence of a precipitating factor** :e.g. infection.
- **Presence of the cause** : uncontrolled hypertension, AS.
- **Terminal cases** of heart failure.

TREATMENT :

- Reassess the cause.
- Removal of mechanical factor : valve replacement.
- Removal of precipitating factor.
- Proper management :
 - Strict bed rest.
 - Salt & even fluid restriction.
 - Proper doses of drugs.
- For terminal cases :
 - Continuous IV positive inotropic therapy.
 - Cardiac resynchronization therapy (CRT)
 - Implantable cardioverter defibrillator (ICD) : for primary prevention of ventricular tachycardia.
 - Mechanical ventilation.
 - Cardiac transplantation : treatment of choice in young patient with severe symptoms & patients with marked low FE (<20%) after applying all antifailure measures.

3

Valvular heart disease

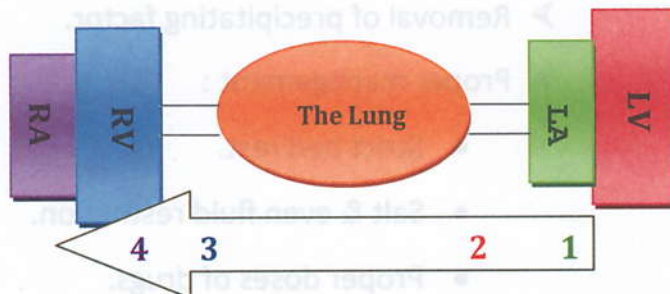
Scheme for left sided valvular heart diseases

Etiology :

MS	AS	MR	AR
	1. Rheumatic (<i>The most common</i>) 2. Congenital. 3. Collagen diseases : SLE, RA. 4. Relative (functional)		
		5. Infective endocarditis. 6. Surgical.	
5-Carey Coomb murmur 6 -Austin Flint murmur.	5- Calcification. 6- IHSS.	7- Mitral prolapse. 8- Papillary muscle dysfunction.	7. Syphilis. 8. Dissecting aorta.

Hemodynamics :

- Left atrial pressure : ↑
- Pulmonary congestion.
- Pulmonary hypertension.
- RSHF. (*late*)
- LSHF (*late*) in all except MS.



In general, any stenosis lead to pressure overload on the upstream cardiac chamber whereas regurgitant lesions cause volume overload.

Clinical picture :

Clinical picture of hemodynamics **PLUS**:

- **MS** ⇒ 4 Stages. (↑LAP , P congestion , P HTN , RSHF)
- **AS** ⇒ Syncope.
- Any **A**orta (AS , AR) ⇒ Angina.
- Any regurge (AR , MR) ⇒ Palpitation , general throbbing.
- AR ⇒ Peripheral signs of AR (9 signs)

Cardiac examination :

Inspection & palpation :

- Apex :

- MS ⇒ Slapping apex.
- AS ⇒ Sustained apex.
- AR, MR ⇒ hyperdynamic apex.

- Pulsation in the 2nd left intercostals space : by appearance of Pulmonary hypertension.

- Signs of ventricular enlargement (late). (**no LVE in MS**).

Percussion : Dullness in the 2nd left intercostals space in a stage of pulmonary HTN.

Auscultation :

i. Normal heart sounds:

- S₁ : ↑ in MS, ↓ in MR.
- S₂ : pulmonary component may be accentuated due to pulmonary HTN (late)

ii. Additional sounds :

- Ejection click (due to P. HTN)
- Gallop (due to heart failure)
- Opening snap : in MS.

iii. Murmur : A M - A M

- Ejection Systolic : **AS**
- Pan systolic : **MR**
- Early diastolic : **AR**
- Mid diastolic : **MS**

AM .. AM

Ahmed Mowafy .. Ahmed Mowafy



Complications : 12

(3 in valve, 3 in LA, 3 in lung, 2 failure & complications of surgery)

1. Calcification.
2. Rheumatic activity.
3. Infective endocarditis. (rare in MS)
4. Arrhythmia e.g. AF in a case of MS, heart block in calcified AS.
5. Thromboembolism : stroke.

6. LA enlargement $\Rightarrow$ compression on :
 - Lung $\Rightarrow$ dyspnea & cough.
 - Esophagus $\Rightarrow$ dysphagia.
 - Left recurrent laryngeal nerve $\Rightarrow$ hoarseness of voice.
7. Pulmonary congestion $\Rightarrow$ hemoptysis & recurrent chest infections.
8. Pulmonary infection.
9. Pulmonary embolism (secondary to DVT)
10. RSHF.
11. LSHF except in MS.
12. Complications of surgery (*artificial valves*) :
 - Mechanical dysfunction.
 - Infective endocarditis.
 - Thromboembolism.
 - Hemolytic anemia.

Investigations :

X ray :

- Chamber enlargement.
- Pulmonary congestion.

ECG :

- Chamber enlargement e.g. LA $\Rightarrow$ P mitrale (m shaped P wave)
- Pulmonary hypertension $\Rightarrow$ P pulmonale (Peaked P wave)

Echo & Doppler echo : (The most important)

- Chamber enlargement .
- Detect the severity of the valve lesion.

Catheterization & angiography :

- Detect the severity.
- Chamber enlargement.

Treatment :

Medical :

- 1- Prophylaxis against IE & rheumatic activity.
- 2- Treatment of complications e.g. HF , AF , infections ...

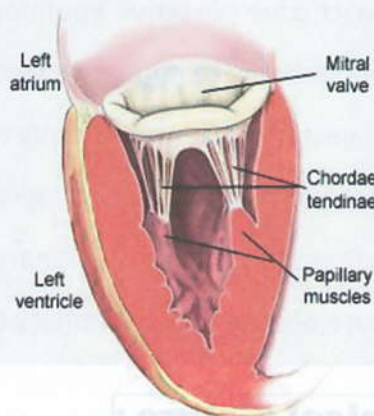
Curative :

- 1- Balloon dilatation (*Percutaneous balloon valvuloplasty*) for stenosis especially pure MS.
- 2- Valvotomy (*commissurotomy*) : for stenosis.
- 3- Valve replacement : Tissue or synthetic valves .

Mitral stenosis

Anatomy of Mitral valve :

- Site : between the LA & LV .
- Surface area : $4 - 5 \text{ cm}^2$, if $< 1 \text{ cm}^2 \Rightarrow$ tight MS.
- Shape of mitral orifice : rounded or oval during diastole & slit like during systole.
- Axis : downward , forward & to the left.
- Components:
 - Fibrous ring.
 - 2 cusps (anteromedial & posterolateral)
 - 2 papillary muscles : arise from the ventricle, to control the cusps movement.
 - Chordae tendinae : arise from papillary muscles to both cusps.



Etiology : (MS is the narrowing of mitral valve orifice $< 2.5 \text{ cm}^2$)

1- Rheumatic heart disease : the commonest cause (99%), more common in female .

Occurs years after the original attack & usually associated with multi valvular lesions.

2- Congenital : Lutembacher's syndrome (ASD + MS), Parachute mitral valve.

3- Relative :

- **Carrey coomb's murmur** : in acute stage of rheumatic fever due to : edema of the cusps $\Rightarrow$ transient narrowing of the mitral valve.
- **Austin-Flint murmur** : murmur of MS in sever AR (The regurged blood during diastole interferes with the opening of mitral valve).
- Conditions of $\uparrow$ blood flow through the mitral valve : VSD , PDA , MR.

Hemodynamics :

4 stages

1- $\uparrow$ LA Pressure with dilatation



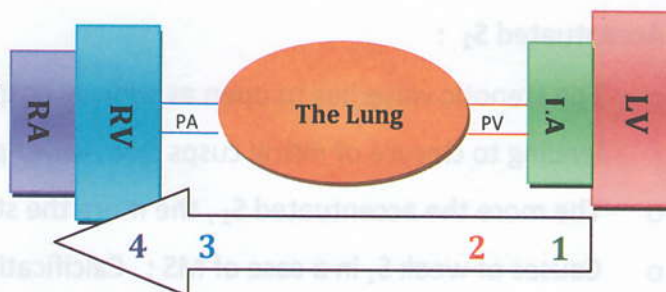
2- back pressure on pulmonary vein (pulmonary congestion)



3- Pulmonary hypertension



4- RSHF



Mechanisms of pulmonary hypertension in MS :

1- Passive pulmonary hypertension.

2- Constrictive (reactive) pulmonary hypertension :

long standing pulmonary congestion $\Rightarrow$ reflex VC of pulmonary arterioles to relieve the congestion but unfortunately with development of pulmonary hypertension.

3- Obstructive pulmonary hypertension :

Long standing VC of pulmonary arteriole $\Rightarrow$ irreversible narrowing of the arterioles.

4- Acute obstructive pulmonary hypertension e.g. pulmonary embolism.

Clinical picture :

- Stage I : asymptomatic (just $\uparrow$ of LA pressure)
- Stage II : manifestations of pulmonary congestion : **dyspnea** ,(*p edema not common*)
- Stage III : manifestations of pulmonary hypertension : **LCOP** , **Malar flush** , giant (a) wave
- Stage IV : manifestations of RSHF : LCOP, systemic congestion.

Cardiac examinations :

Inspection & palpation :

- Slapping apex : weak impulse (due to $\downarrow$ LV filling) with palpable S_1 (due to accentuated S_1)
- Apical diastolic thrill.
- Stage III : Pulsation & systolic thrill in the 2nd left intercostals space with palpable S_2 .

Percussion : Stage III : Dullness in the 2nd left intercostals space .

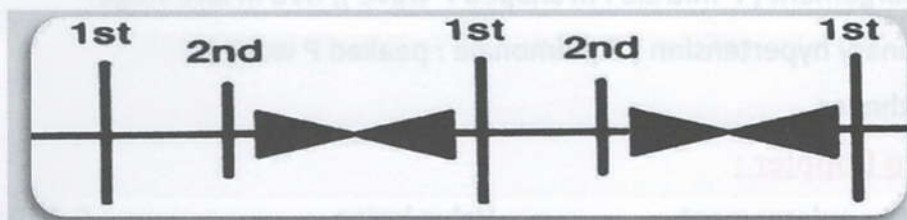
Auscultation :

Normal heart sounds :

- **Accentuated S_1 :**
 - The stenotic valve has to open as wide as possible to overcome this stenosis , leading to closure of mitral cusps from lower position $\Rightarrow \uparrow S_1$.
 - The more the accentuated S_1 , the more the stenosis.
 - Causes of weak S_1 in a case of MS : Calcification & Associated MR.
- **S_2 :** normal , may be accentuated in **stage III** (pulmonary hypertension)

Additional sounds :**1. Opening snap :**

- Sharp snapping sound following S₂ due to sudden opening of rigid cusps.
- The closer the snap to S₂, the more the stenosis.
- Calcification causes disappearance of opening snap.

2. Ejection click : in stage III (Pulmonary hypertension)**3. Gallop on tricuspid area :** in stage IV (RSHF)**Murmur :**comment on : **SCRIPT**

- **Site** : best heard at or inside the apex.
- **Character** : rumbling , low pitched murmur.
- **Relation to respiration & position** : ↑ with expiration & ↑ in left lateral position.
 - ✓ Left sided heart murmurs are louder on expiration .
 - ✓ Right sided heart murmurs are louder on inspiration .
- **Intensity** : pre systolic accentuation due to atrial contraction.
Pre systolic murmur is absent in AF.
- **Propagation** : No propagation (localized)
- **Timing** : **Mid diastolic , pre systolic.**

Silent mitral stenosis : (↓ blood flow through mitral valve ⇔ no murmur)

- 1- Associated ASD (Lutembacher's syndrome)
- 2- Low LA pressure : sever pulmonary hypertension , RSHF.
- 3- High LV pressure : LSHF.

Stage III : Ejection systolic murmur of pulmonary hypertension.

Stage IV : Pansystolic murmur of relative TR.

Investigation :**X ray :**

- LA enlargement (lateral view with barium)
- Pulmonary congestion.
- Dilated pulmonary artery.
- RVE.
- Calcification of mitral valve.

ECG :

- LA enlargement (P mitrale : m shaped P wave), RVE in late stage.
- Pulmonary hypertension (P pulmonale : peaked P wave)
- Arrhythmias.

Echo & echo Doppler :

- Chamber enlargement .
- Valve lesion.
- Calcification.

Catheterization & angiography:

- Chamber enlargement.
- Mitral stenosis index = $\text{COP/LAP} \times 100 = 5/5 \times 100 = 100\%$ (< 25 % is tight MS)

Complications : see scheme.**Treatment :****Medical :**

- 1- Prophylaxis against IE & rheumatic activity.
- 2- Treatment of complications e.g. HF , AF , infections ...

Surgical : Not very early , not very late

- 1- Balloon dilatation: In pure MS (no calcification , not combined with MR , not severe)
- 2- Valvotomy (commissurotomy) .
- 3- Valve replacement : Tissue or synthetic valves.

Indications of valve replacement :

- Calcification
- Associated MR
- Tight MS (MS Index < 25 % , surface area < 1cm^2 or severe manifestations)
- recurrent stenosis after balloon dilatation or valvotomy .

Mitral Regurge

(Mitral Insufficiency)

Etiology :

1. Rheumatic (*the commonest*)
2. Congenital.
3. Collagen diseases : SLE, RA ...
4. Infective endocarditis.
5. Surgical.
6. **Mitral valve prolapse.**
7. **Papillary muscle dysfunction** : MI, Marfan syndrome.
8. Functional (relative) : dilatation of mitral ring due to dilatation of LV e.g. LSHF , AR.

Hemodynamics :

- **During systole** : A part of blood regurgitates from LV to LA leading to LA dilatation .
- **During diastole** : ↑ blood flow through the mitral valve ⇒ ↑ volume load on LV ⇒ LV enlargement then failure.

In acute MR : as in myocardial infarction & IE , the LA has no time to dilate and accommodate the regurgitant blood ⇒ great ↑ of LAP ⇒ rapid pulmonary congestion then pulmonary edema & can lead to cardiogenic shock.

Clinical picture :

- 1- Asymptomatic for many years in mild cases.
- 2- **Palpitation** & general throbbing due to LV volume overload.
- 3- Manifestations of LSHF (late)
- 4- Manifestations of pulmonary congestion then pulmonary hypertension , RSHF later.
- 5- Acute pulmonary edema in a case of acute MR.

Cardiac examination :

Inspection & palpation :

- LVE .
- Hyperdynamic apex (*forcible , non sustained apex*).
- Systolic thrill over the apex.

Auscultation :**Normal heart sounds :**

- **S₁** : weak (due to weak closure of mitral valve & masking by pansystolic murmur of MR)
- **S₂** : may be accentuated with development of pulmonary hypertension.

Additional sounds:

- With HF ⇒ Gallop.
- With pulmonary hypertension ⇒ Ejection click.

Murmur :**- Murmur of MR :**

- **Site** : best heard at the apex.
- **Character** : blowing.
- **Relation to respiration & position** : ↑ with expiration & ↑ in left lateral position.
- **Propagation** : to axilla (except in posterior leaflet regurge radiate to the base of heart)
- **Timing** : **Pansystolic murmur**. (plateau)
- Relative MS : mid diastolic murmur due to excess blood flow across the mitral valve.

Complications : *see scheme.***Investigations :**

- **X ray** : As in MS + **LV dilatation**.
- **ECG** : Chamber enlargement, arrhythmias.
- **Echo** : - Chamber enlargement - Valve lesion.
- **Catheterization** : - Chamber enlargement - Valve lesion .

Treatment :

- Medical : *see scheme* .
- Surgical : valve replacement or mitral valve repair.

Mitral valve prolapse

Definition : Prolapse of one or both cusps of mitral valve into LA during systole.

Etiology :

1. Idiopathic : in most cases , more common in young female.
2. Connective tissue diseases :
 - Marfan syndrome.
 - Ehlers-Danlos syndrome.
 - SLE.
 - Polyarthritis nodosa.
3. Muscle disorders : Duchenne myopathy, Myotonia dystrophy.
4. Congenital heart diseases : e.g. ASD
5. Acquired heart diseases : MI, post mitral valve surgery.

C/P :

- Asymptomatic in most cases.
- Atypical chest pain is the most common symptom. Usually it is left submammary & stabbing. Sometimes it is severe substernal aching pain.
- Palpitation : due to abnormal ventricular contraction & arrhythmias.
- Fainting & syncope.
- Sudden death due to fatal ventricular arrhythmias (very rare).

Cardiac examination :

- The most common sign is a mid-systolic click, which is produced by the sudden prolapse of the valve & the tension of the chordate tendineae.
- This may be followed by a late systolic murmur due to some regurgitation.
- With more regurgitation, the murmur becomes pansystolic.

Investigations : Echo is diagnostic

Treatment :

- Reassurance.
- Prophylaxis against SBE ?
- β blocker e.g. propranolol.
- valve replacement in severe cases.

Aortic Stenosis

Anatomy :

- 3 semilunar cusps attached to a fibrous valve ring .
- In about 1 % of individuals , only 2 cusps are present (Bicuspid aortic valve).
- Surface area is about 3 cm² .

Etiology :

- 1- Rheumatic fever.
- 2- Congenital : it may be valvular , subvalvular or supra- valvular.
- 3- Calcifications.
- 4- Hypertrophic cardiomyopathy : (*Idiopathic Hypertrophic Subaortic Stenosis - IHSS*)
- 5- Relative :
 - ↑ blood flow across the aortic valve : AR.
 - Dilatation of aorta : Hypertension , atherosclerosis .

Hemodynamics :

During systole , there is obstruction of LV outflow results in :

- LCOP.
- Pressure overload on LV leading to LV hypertrophy then failure.

Clinical picture :

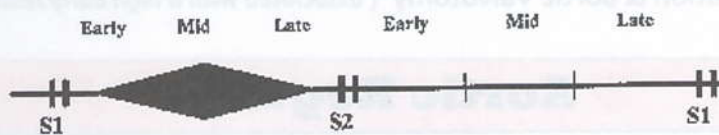
- Asymptomatic in mild cases. ➤ Manifestations of LCOP.
- **Syncope** : especially Exertional due to low fixed COP.
- **Angina** : Due to :
 - LCOP ⇒ ↓ coronary blood flow.
 - LV hypertrophy ⇒ ↑ O₂ demand.
 - Associated atherosclerosis or AR.
- Manifestations of LSHF.

Cardiac examinations :

- LVE.
- Sustained apex : (*forcible ,sustained apex*)
- Systolic thrill over 2nd right intercostal space (A₁) & propagated to apex & neck.

Auscultation :

- Weak S_2 with closed , single or paradoxical splitting (*delayed aortic component*)
- Additional sounds :
 - Ejection click due to opening of rigid aortic cusps , disappears with calcification.
 - gallop due to LSHF.
 - S_4 : due to pressure overload on the LV.

Murmur :**- Murmur of AS :**

- **Site** : maximum over A_1 area (2nd right intercostal space).
 - **Character** : Harsh but may be soft in relative AS.
 - **Relation to respiration & position** : ↑ with expiration & with leaning forward.
 - **Propagation** : neck (carotid arteries) & apex.
 - **Timing** : Ejection (mid) systolic murmur (*diamond shaped* , crescendo decrescendo)
- Murmur of functional MR (*due to dilated LV*) : pansystolic murmur on the apex .

Complications : see scheme**Investigation :**

- **X ray** :
 - LVE.
 - Post stenotic dilatation (in valvular type)
 - Pulmonary congestion.
 - Calcification.
- **ECG** : LVE.
- **Echo** : Detects the severity of valve lesion ($< 0.8 \text{ cm}^2 \Rightarrow$ severe AS)
Chamber enlargement.
- **Catheterization** : Detects the severity (*it can measure the pressure gradient across aortic valve*)

Treatment :

Medical : same as scheme *plus* , β blocker for angina.

Surgical :➤ Aortic valve replacement :

- Indication : - Valve area $< 0.8 \text{ cm}^2$
 - Systolic pressure gradient across the aortic valve $> 50 \text{ mmHg}$.
 - Severe symptoms.

➤ Balloon dilatation & aortic Valvotomy (associated with a high early restenosis rate)

Aortic Regurge**Etiology :**

1- Rheumatic fever.

2- Congenital.

3- Infective endocarditis.

4- Surgical.

5- Dilatation of the ascending aorta :

- Syphilis.
- Marfan syndrome.
- Severe hypertension.
- Aortic dissection.
- Ankylosing spondylitis.

Hemodynamics :

During diastole : regurgitation of blood from the aorta to the LV leading to : 🐼

- Volume overload on the LV $\Rightarrow$ LVE the failure.
- $\downarrow$ coronary blood flow $\Rightarrow$ Angina.
- $\uparrow$ blood in LV $\Rightarrow$ $\uparrow$ LV contraction $\Rightarrow$ $\uparrow$ stroke volume $\Rightarrow$ $\uparrow$ systolic pressure .

This high systolic BP is compensated by peripheral VD which (together with regurgitation) will decrease the diastolic BP. So in AR :

$\uparrow$ Systolic BP : due to high stroke volume (high COP).

$\downarrow$ Diastolic BP : due to peripheral VD & regurgitation of blood during diastole.

Clinical picture :

- 1- Asymptomatic in mild cases.
- 2- **General throbbing** : due to ↑ arterial pulsation.
- 3- **Palpitation** : due to forcible LV contraction.
- 4- **Angina** : there are 2 types
 - **Classic angina** : due to :
 - ↓ Diastolic BP ⇒ ↓ coronary blood flow.
 - LVE ⇒ ↑ O₂ demand.
 - **Angina of Lewis** : Nocturnal , prolonged angina & associated with autonomic disturbances (sweating , tachycardia)
- 5- Manifestations of LSHF : Pulmonary congestion & LCOP.

Peripheral signs of AR : (due to big pulse volume) *3 in neck , 3 in UL , 3 in LL*

Head & neck :

- De Musset sign : nodding of the head.
- Corrigan's sign : Marked visible carotid pulsation.
- Systolic thrill over the carotid artery.

Upper limb :

- BP : ↑ systolic & ↓ diastolic BP.
- Pulse : Water hammer pulse.
- Capillary pulsations : pressing on the nail tip ⇒ moving red line.

Lower limb :

- Pistol shots : systolic femoral sound due to sudden distension of collapsed artery.
- Duroziez's sign : systolic & diastolic murmur over the femoral artery if slight pressure is applied to it by the stethoscope.
- Hill's sign : The difference between systolic BP in LL & UL > 50 mmHg.
(Normally SBP in LL > UL by 10 - 20 mmHg)

AB : AR with minimal peripheral signs :

- Mild AR.
- ↓ systolic BP : MS , AS.
- ↑ Diastolic BP : Systemic hypertension.

Cardiac examination :

- LVE .
- Hyperdynamic apex.
- No thrill over the aortic area in isolated AR.

Auscultation :

- S₂ : usually normal in isolated AR.
- Additional sounds : gallop on the mitral area.

Murmur :**i. Murmur of AR :**

- **Site** : - Maximum over the 3rd left intercostals space (A₂ area)
 - In syphilitic AR : maximum over A₁ (2nd right intercostals space.
- **Character** : Soft blowing , decrescendo .
- **Relation to respiration & position** : ↑ with expiration & with leaning forward.
- **Propagation** : To apex & left sterna border.
- **Timing** : Early diastolic.

ii. Murmur of relative AS : ejection systolic murmur (soft)**Murmurs over the mitral area (apex) in case of AR :**

- 1- Mid diastolic murmur of relative MS (*Austin - Flint murmur*)
- 2- Pansystolic murmur of relative MR.
- 3- Propagation of ejection systolic murmur of relative or combined AS.
- 4- Propagation of early diastolic murmur of AR itself.

Complications :

see scheme .

Investigations :

- **X ray** : LVE & dilated aorta (Boot - shaped heart)
- **ECG** : LVE.
- **Echo** : LVE , detects the severity of the valve lesion.
- **Catheterization** : LVE , detects the severity of the valve lesion.

Treatment :

Medical : As scheme.

Surgical : Valve replacement in severe organic cases with LV dysfunction.

	Rheumatic AR	Syphilitic AR
Age	20 - 40 years	> 40 years
History	Of rheumatic fever	Of syphilis
Valvular lesion	yes	no
Angina	Less common.	More common.
S ₂	Usually normal	↑
Murmur (<i>maximum intensity</i>)	Over A ₂	Over A ₁
X ray	Calcification	Aortic aneurysm.

Tricuspid Stenosis

Etiology :

It's usually rheumatic in origin & usually associated with mitral or aortic valve diseases.

Hemodynamics : obstruction of tricuspid valve leading to :

- ↑ RA pressure ⇒ RA enlargement & systemic congestion.
- ↓ RV filling ⇒ ↓ COP.

Clinical picture:

- Symptoms of LCOP.
- Symptoms of associated lesions e.g. MS
- Symptoms of systemic congestion.

NB : TS ⇒ ↓ symptoms of MS due to restriction of pulmonary flow.

General signs :

- LCOP.
- Neck vein : Giant (a) wave.
- Systemic congestion.

Cardiac sign :

- RA & RV enlargement.
- mid diastolic presystolic murmur at lower left sternal border, increases by inspiration.

Investigation :

- X ray , ECG : RA & RV enlargement.
- Echo & Catheterization : diagnostic.

Treatment : Valve replacement.

Tricuspid Regurge

Etiology : TR is usually functional resulting from RVE $\Rightarrow$ dilatation of tricuspid ring.

Hemodynamics : During systole, part of blood regurgitates from RV to RA causing:

- $\uparrow$ RA pressure $\Rightarrow$ RA enlargement & systemic congestion.
- LCOP.
- RVE then failure.

Clinical picture :

Symptoms : - of the cause. - Systemic congestion. - LCOP.

General signs :

➤ **Signs of systemic congestion :**

- Congested pulsating neck vein with systolic expansion.
- Enlarged tender pulsating liver with mild jaundice.
- Ascites before edema LL.

➤ **Signs of LCOP :** cold hand , weak pulse , $\downarrow$ SBP , peripheral cyanosis.

Mild jaundice (liver congestion) with peripheral cyanosis (LCOP) $\Rightarrow$ **Cyano - ictric face.**

Cardiac signs :

- RA & RV enlargement
- Systolic thrill over tricuspid area.
- **Murmur :**
 - Pansystolic murmur.
 - Increased by inspiration.
 - Maximum over tricuspid area & propagated to the apex but not to the axilla.

Complications : - RSHF - IE - Cardiac cirrhosis.

Investigations :

- X ray & ECG : RA & RV enlargement.
- Echo & catheterization : Diagnostic.

Treatment :

- Treatment of RSHF.
- Valve replacement.

DD of systolic murmurs :

1. AS
2. PS
3. MR
4. TR
5. VSD
6. PDA
7. Coarctation of aorta

Over the apex :

- MR
- Propagated from other area: all of the above 7 lesions may propagate to the apex.

Over the base :

- All of the above except MR & TR.
- Posterior leaflet MR may propagate to the base.

DD of diastolic murmurs :

1. AR
2. PR
3. MS
4. TS
5. PDA
6. Coarctation of aorta

Over the apex :

- MS : either organic or relative.
- Propagated from other area : AR , PR

Over the base :

- AR , PR , PDA
- Coarctation of aorta (due to collaterals)

4

Congenital heart disease

Criteria to suspect Congenital HD:

- Cyanosis since birth.
- Murmur since birth.
- No history of rheumatic fever.
- Recurrent chest infection.
- Hypertensive child.
- +ve family history.
- Associated congenital anomalies.

Classification:

Cyanotic :

- Fallot's tetralogy (F₄)
- Fallot's triology (F₃)
- Eisenmenger's syndrome.
- Tricuspid atresia.
- Transposition of the great arteries.

Fallot's pentalogy (F₅) : F₄ + ASD

Acyanotic :

- With RV enlargement : ASD , PS .
- With LV enlargement : AS , PDA , Coarctation of aorta.
- With biventricular enlargement : VSD .
- With NO ventricular enlargement : : Any mild lesion , Dextrocardia .

ATRIAL SEPTAL DEFECT (ASD)

Anatomy :

- There is an abnormal opening between the two atria, producing left to right shunt.
 - High ASD (ostium secundum) : the most common.
 - Low ASD (ostium premium) : may be associated with Mitral valve disease (Lutembacher's syndrome) MCQ

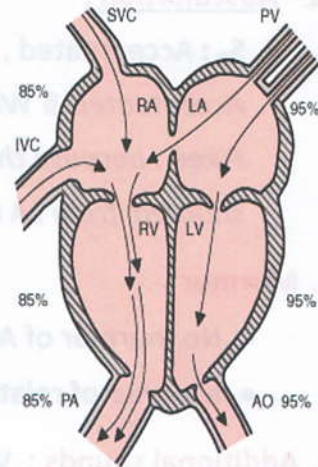
Hemodynamics :

As the pressure in LA is higher than in RA , blood is shunted from LA $\Rightarrow$ RA $\Rightarrow$ RV (causing **RVE**) $\Rightarrow$ PA (causing its dilatation) $\Rightarrow$ Lung ($\uparrow$ blood flow through pulmonary arterioles causing **lung plethora** & then pulmonary hypertension) $\Rightarrow$ The blood from lung comes back to LA & the cycle is repeated .

- Notice that blood passing from LA to LV will be less than normal $\Rightarrow$ **LCOP**.

So, in ASD there are :

- Lung plethora.
- LCOP.
- RVE.



Notice that O₂ level is high in the RV & PA (N : 75%)

Clinical picture :**Symptoms :**

- 1- Asymptomatic in mild cases or in early life.
- 2- Symptoms of hemodynamics :
 - Symptoms of lung plethora : Exertional dyspnea , **recurrent chest infection...**
 - Symptoms of LCOP .
- 3- Symptoms of complication.
- 4- Symptoms of associated congenital anomalies : Secundum ASD may be associated with tri-phalangeal thumb & radial abnormalities.

MCQ**Signs :**

- 1- No signs in mild cases.
- 2- Signs of hemodynamics :
 - Signs of lung plethora.
 - Signs of LCOP.
- 3- Signs of complication.

4- **Neck vein** : No giant (a) wave inspite of pulmonary hypertension ?

Cardiac examination :

1- RVE.

2- Auscultation :

i. S_2 : Accentuated , wide **fixed** splitting S_2

- *Accentuated & Wide splitting : due to pulmonary hypertension.*
- ***Fixed** : because the $\uparrow$ VR to the RA during inspiration is compensated by $\downarrow$ blood shunted from LA to RA .*

ii. Murmur :

- No murmur of ASD itself because of low pressure gradients between the 2 atria.
- Murmur of relative TS & PS may be heard.

iii. Additional sounds : Ventricular Gallop due to RSHF.

Complication :

- 1- RSHF.
- 2- Paradoxical embolism e.g. stroke.
- 3- Eisenmenger's syndrome: cyanosis with shunt reversal.
- 4- Infective endocarditis : rare due to low pressure gradient.
- 5- Arrhythmia : AF

Investigations :

- **X ray** : RVE , dilated pulmonary artery , lung plethora.
- **ECG** : RBBB in most cases , RVE.
- **Echo** : RVE , detect the defect.
- **Catheterization :**
 - Detect the defect : the catheter may pass through ASD.
 - $\uparrow$ pressure in the right side of the heart.
 - $\uparrow$ O_2 level in RA in comparison to superior & inferior vena cava.

Treatment :

- Closure of the defect : must be done before reversal of the shunt.
- Treatment of complications.

PULMONARY STENOSIS (PS)

Anatomy :

- Valvular : the most common type (80 %) .
- Subvalvular (Infundibular) .
- Supravalvular : rare.

Hemodynamics :

PS $\Rightarrow$ $\uparrow$ the resistance (*afterload*) against the RV leading to :

- LCOP.
- RVE then failure.

Clinical picture :

Symptoms :

- Asymptomatic in mild cases.
- Symptoms of hemodynamics : LCOP , RSHF.

Signs :

General :

- Signs of LCOP.
- Signs of RSHF.
- Giant (a) wave.

Cardiac :

- RVE.
- Systolic thrill on pulmonary area.
- Auscultation :
 - S_2 : weak pulmonary component of S_2 with wide splitting.
 - Additional sounds : Ejection click in valvular type , S_4 on tricuspid area.
 - Murmur : ejection systolic murmur on pulmonary area.

Complications :

- RSHF.
- Infective endocarditis.
- TB due to lung oligemia.

Investigations :

- **X ray** : RVE , Lung oligemia , Post stenotic dilatation in valvular type.
- **ECG** : P pulmonale, RVE.
- **Echo** : Diagnostic.
- **Catheterization**: Diagnostic.

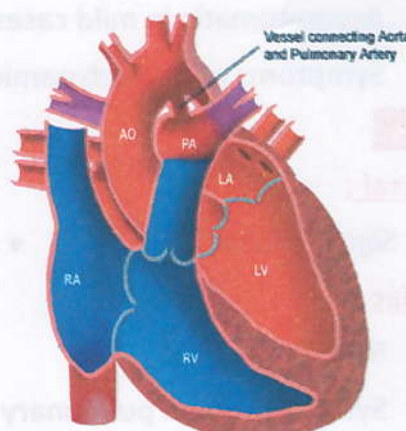
detects the pressure gradient across the pulmonary valve : if $>50 \Rightarrow$ severe PS.

Treatment :

- Prophylaxis against infective endocarditis
- Treatment of RSHF
- Surgical : in severe PS
 - Valvular type : valvotomy or replacement.
 - Subvalvular type : resection of infundibulum.

PATENT DUCTUS ARTERIOSUS(PDA)**Anatomy :**

- Persistence of ductus arteriosus between the left pulmonary artery & the aorta just distal to the left subclavian artery.
- PDA is normal during fetal life & it closes soon or shortly after birth.
- PDA is common in premature babies , particularly female infants.

**Hemodynamics :**

The aortic pressure (120/80 mmHg) is higher than pulmonary pressure (20/10 mmHg) in both systole & diastole so the blood is shunted from aorta to PA in both systole & diastole leading to :

- ↑ blood flow to pulmonary arteries (**lung plethora**) ⇒ ↑ blood flow to LA ⇒ to LV causing **LVE (later failure)** ⇒ to the aorta causing high COP & high systolic BP.
- The escape of blood from the aorta to the PA causes low diastolic BP.
- High SBP & low DBP ⇒ **hyperdynamic circulation**
- Later on, pulmonary hypertension & reversal of the shunt occur (**Eisenmenger's syndrome**)

Clinical picture :**Symptoms :**

- 1- No symptoms in mild cases.
- 2- **Symptoms of hemodynamics :**
 - Symptoms of lung plethora.
 - Symptoms of hyperdynamic circulation : palpitation & general throbbing.
- 3- Symptoms of complications.
- 4- Symptoms of other congenital anomalies.

Signs :

- 1- No signs in mild cases .
- 2- **Signs of hemodynamics :**
 - Signs of lung plethora.
 - Signs of hyperdynamic circulation : the same as peripheral signs of AR. (9)
- 3- Signs of complications.
- 4- **Neck vein :** Giant (a) wave due to pulmonary hypertension.

Cardiac examination :

1. LVE.
2. Continuous thrill over left infraclavicular area (*site of DA*).
3. **Auscultation :**
 - S_2 : Accentuated, reversed splitting S_2 (due to delayed evacuation of LV).
 - Murmur : Continuous "*machinery*" murmur over left infraclavicular area.

DD of continuous murmur :

- 1- **A**rterio-venous fistula.
- 2- **P**DA.
- 3- **C**oarctation of aorta.
- 4- **D**ouble mitral (combined MS & MR).

Complications :

- 1- LSHF.
- 2- Paradoxical embolism e.g. stroke.
- 3- Infective endocarditis.
- 4- Eisenmenger's syndrome $\Rightarrow$ differential cyanosis (*cyanosis only in LL*) because the reversed cyanotic blood enter aorta distal to subclavian artery.
- 5- Arrhythmia.

Investigation :

X ray : - Dilatation of aorta , PA , LA & LV. - Lung plethora.

ECG : LVE.

Echo : show chamber dilatation.

Catheterization :

- Detect the defect : the catheter may pass through PDA.
- $\uparrow$ Pulmonary pressure.
- $\uparrow$ O₂ level in PA in comparison to RV.

Treatment :**Medical :**

- Prophylaxis against infective endocarditis.
- Treatment of complications
- Medical closure of the duct : Indomethacin.

Surgical : Closure of the duct.

COARCTATION OF AORTA

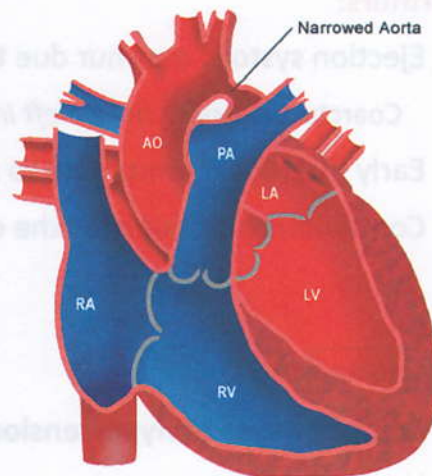
Anatomy :

- Congenital narrowing of a part of aorta usually distal to the left subclavian artery.
- Associated congenital anomalies :
Bicuspid aortic valve (AS, AR) , PDA , VSD , Congenital aneurism of Circle of Willis ,
Turner's syndrome.

Hemodynamics :

Narrowing of a part of aorta causes : 

- ↑ BP in the proximal part (*before the narrowing*)
- ↓ BP in the distal part (*after the narrowing*)
- Development of collaterals between the proximal & distal part.



Clinical picture :

Symptoms :

1- Asymptomatic in mild cases.

2- Symptoms of hemodynamics :

- ↑ BP in the upper half ⇒ Symptoms of hypertension e.g. headache , epistaxis ..
- ↓ BP in the lower half ⇒ Fatigue & Intermittent claudication of the LL.
- Collaterals ⇒ Pain around left shoulder.

3- Symptoms of complications.

4- Symptoms of other congenital anomalies e.g. AS , AR , PDA

Signs :

1- No signs in mild cases.

2- Signs of hemodynamics :

- ↑ BP in arms, prominent carotid pulsation.
- ↓ BP in legs , weak pulsations of LL e.g. dorsalis pedis.
- Collaterals ⇒ may be seen in interscapular area (Suzman's sign).

- 3- Signs of complications.
- 4- Signs of other congenital anomalies.

Cardiac examination :

1. LV hypertrophy.
2. Auscultation :
 - Accentuated S₂
 - **Murmurs:**
 - Ejection systolic murmur due to :
Coarctation itself (*below left infraclavicular area*) , Associated AS ,Hypertension.
 - Early diastolic murmur due to associated AR.
 - Continuous murmur over the collaterals.

Complications :

- 1- **Complications of hypertension** e.g. cerebral hemorrhage
- 2- Heart failure.
- 3- Infective endocarditis.

Investigations :

- 1- X ray :
 - LVE.
 - Rosler's sign : Rib notching (3-8) due to erosion by collaterals.
- 2- ECG : LVE .
- 3- Echo : LVE , can detect the coarctation .
- 4- Catheterization & aortography : can detect the site & severity of the coarctation.

Treatment :

- Medical : prophylaxis against IE & treatment of the complications.
- Surgical repair : in early childhood to avoid persistent hypertension.

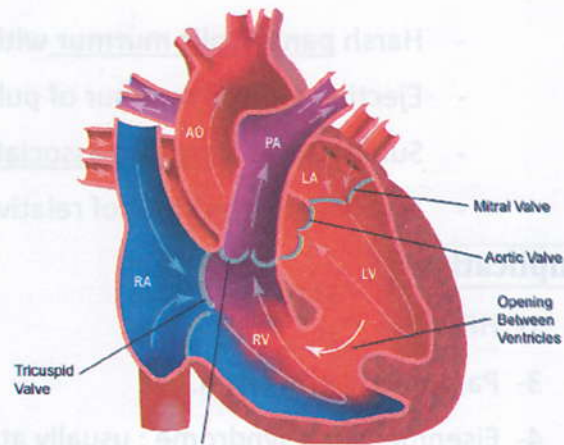
VENTRICULAR SEPTAL DEFECT (VSD)

Anatomy :

- There is an abnormal opening between the two ventricles, producing left to right shunt.
- There are 2 types :
 - Big membranous type : occurs in the membranous part of the interventricular septum.
 - Small muscular type (*Roger's disease*) : occurs in muscular part of interventricular septum , it's hemodynamically insignificant & more than 90% of cases close spontaneously.

Hemodynamics :

- The pressure in LV is 120 / 0 mmHg.
- The pressure in RV is 25 / 0 mmHg.
- So , the blood is shunted from LV to RV**
- during systole only leading to :**
- The shunted blood to the RV causes **RVE**
 - ⇒ ↑ blood flow to pulmonary arteries
 - (lung plethora & pulmonary hypertension)**
 - ⇒ ↑ blood flow to LA ⇒ to LV causing **LVE**
 - (later failure)**
- Notice that blood passing from LV to aorta will be less than normal ⇒ **LCOP**.



So, in VSD there are :

- Lung plethora.
- LCOP
- Biventricular enlargement.

Clinical picture :

Symptoms :

1. Asymptomatic in mild cases & in Roger's disease.
2. **Symptoms of hemodynamics :**
 - Symptoms of lung plethora : Exertional dyspnea , recurrent chest infection...
 - Symptoms of LCOP.
3. Symptoms of the complications.
4. Symptoms of other congenital anomalies.

Signs :

1. No signs in mild cases.
2. **Signs of hemodynamics** : Lung plethora & LCOP.
3. Signs of complications.
4. **Neck vein** : Giant (a) wave.

Cardiac examination :

1. Biventricular enlargement with hyperdynamic apex.
2. Auscultation :
 - S₂ : Accentuated pulmonary component , wide splitting.
 - **Murmur** :
 - Harsh **pansystolic murmur** with thrill over the 3rd, 4th intercostal spaces.
 - Ejection systolic murmur of pulmonary hypertension.
 - Sub-aortic VSD may be associated with AR
 - Mid diastolic murmur of relative MS (↑ blood flow across the mitral valve)

Complication :

- 1- HF.
- 2- Infective endocarditis.
- 3- Paradoxical embolism.
- 4- Eisenmenger's syndrome : usually at 2nd - 3rd decade.

Investigations :

1. **X ray** : Biventricular enlargement , lung plethora.
2. **ECG** : Biventricular enlargement.
3. **Echo** : Biventricular enlargement , diagnosis of anomaly.
4. **Catheterization** :
 - Detect the defect : the catheter may pass through VSD.
 - ↑ pressure in the RV & PA.
 - ↑ O₂ level in RV in comparison to RA.

Treatment :

- Prophylaxis against IE & treatment of complications.
- Surgical closure of large defect.

TETRALOGY OF FALLOT (F₄)

Anatomy :

Slight deviation of the upper part of interventricular septum to the right leading to:

- 1- PS (*subvalvular*)
- 2- Mild RVE.
- 3- VSD (*not significant*)
- 4- Overriding of Aorta.

Hemodynamics :

- 1- **PS** : deoxygenated blood passes to aorta ⇒ p. oligemia & central cyanosis.
- 2- Mild RVE : because RV has 2 ways: stenosed PA & wide aorta.
- 3- Overriding of aorta : it receives blood from both ventricles ⇒ central cyanosis.
- 4- VSD : not significant (*silent*) despite big VSD because the pressures in both ventricles are equal.

Clinical picture :

Symptoms :

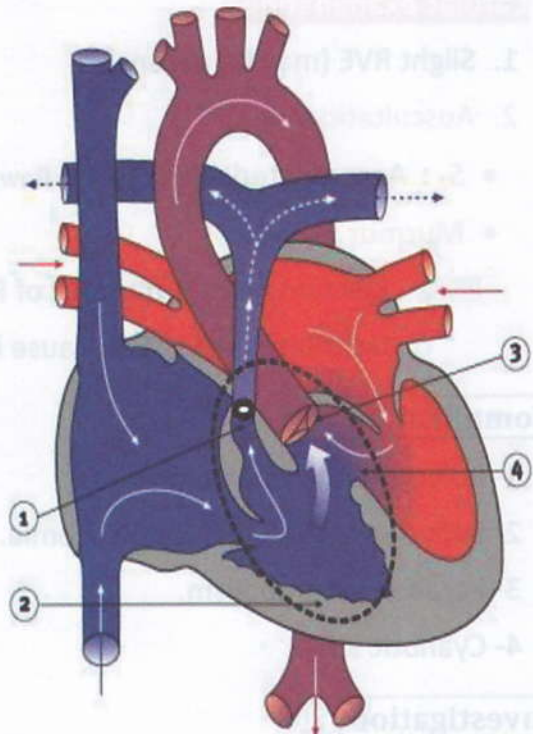
- 1- **C**entral cyanosis since birth or shortly after.
- 2- **C**entral dyspnea. (hypoxia ⇒ ↑ respiratory center)
- 3- **C**lubbing.

4- **Cyanotic spell** : (↑ cyanosis with exertion)

Exertion ⇒ sympathetic stimulation ⇒ spasm of subvalvular tissue (*infundibulum*) of the RV ⇒ severe PS ⇒ ↑ deoxygenated blood flow to aorta ⇒ ↑ cyanosis.

C/P :

1. ↑ **C**yanosis
2. **C**onvulsion
3. **C**ardiac arrest .
4. **S**quatting position ⇒ kinking of femoral artery ⇒ ↑ resistance of the aorta ⇒ ↓ blood flow from RV to aorta ⇒ more blood to the lung ⇒ ↓ cyanosis & dyspnea



Signs :

- 1- Central cyanosis
- 2- Clubbing
- 3- Stunted (delayed) growth.
- 4- **Neck vein** : dominant (a) wave.

Cardiac examination :

1. Slight RVE (may be absent)
2. **Auscultation** :
 - **S₂** : Accentuated(due to ↑aortic flow), **Single**(pulmonary component is very weak to be heard)
 - **Murmur** :
 - Ejection systolic murmur of PS.
 - No murmur of VSD because it is not significant (*silent VSD*).

Complication :

- 1- Polycythemia due to hypoxia.
- 2- Pulmonary TB due to lung oligemia.
- 3- Paradoxical embolism.
- 4- Cyanotic spell.

Investigations :

- 1- **X ray** : Boot shaped heart : narrow base with elevated apex.
Pulmonary oligemia.
- 2- **ECG** : RVE.
- 3- **Echo** :diagnostic.
- 4- **Catheterization** : diagnostic.

Treatment :

- 1- Treatment of cyanotic spell : Squatting position , O₂ , β blocker.
- 2- Prophylaxis against infective endocarditis.
- 3- **Blalock Taussig** operation : acquired PDA.
- 4- Surgical total correction.

TRIOLOGY OF FALLOT (F₃)

1. PS (*valvular*)
2. Marked RVE.
3. ASD.

EISENMENGER'S SYNDROME**Definition :**

It is a condition in which a left-to-right shunt in the heart causes pulmonary hypertension, which in turn ,causes increased pressure in the right side of the heart and **reversal of the shunt into a right-to-left shunt.**

Etiology :

- VSD.
- PDA.
- ASD.

Eisenmenger complex was applied to patients with reversal of shunt in a case of VSD by Dr. *Victor Eisenmenger* in 1897 but the definition was extended by Dr. *Paul Wood* to include shunts at any level VSD, ASD, PDA ,..

Clinical picture :

- 1- History of congenital heart disease : VSD , PDA , ASD.
- 2- Pulmonary infection & hemoptysis.
- 3- C/P of pulmonary hypertension .
- 4- C/P of RSHF.
- 5- Decrease of the original murmur of the shunt due to low pressure gradient.

Treatment :

- Prevention is best.
- Closure of the defect is contraindicated as it increases the pressure in the right side of the heart.
- Symptomatic treatment e.g. HF.
- Heart lung transplantation.

TRANSPOSITION OF THE GREAT VESSELS (TGA)

- The position of the aorta & the PA are reversed, this leads to separate 2 circuits :
- The aorta arises from the RV ,so most of the blood returning to the heart from the body is pumped back out through the aorta without going to the lung ⇒ central cyanosis.
- The PA arises from the LV ,so the blood returning from the lungs goes back to the lungs again.
- To maintain life , an associated ASD , VSD , PDA ,PS should exist.
- Treatment : Keep the PDA by Prostaglandin E1 , surgical correction.

DEXTROCARDIA

Deviation of the heart to the right , it may be congenital or acquired.

- ***Situs inversus totalis*** : mirror like transposition of the heart & all other viscera.
- ***Isolated dextrocardia*** : mirror like transposition of the heart only.
- ***Dextroversion***: the heart is displaced to the right (RV remains to the right & LV to the left)
- ***Acquired dextrocardia***: acquired displacement of the heart to the right e.g. fibrosis.

5

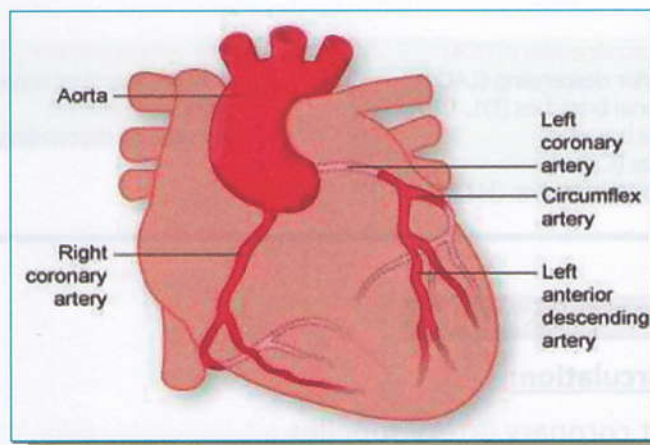
Ischemic heart disease

Synonyms :

Coronary artery disease, coronary ischemia, coronary atherosclerosis.

Anatomy of coronary arteries :

There are two coronary arteries – left & right – originate from the root of ascending aorta.



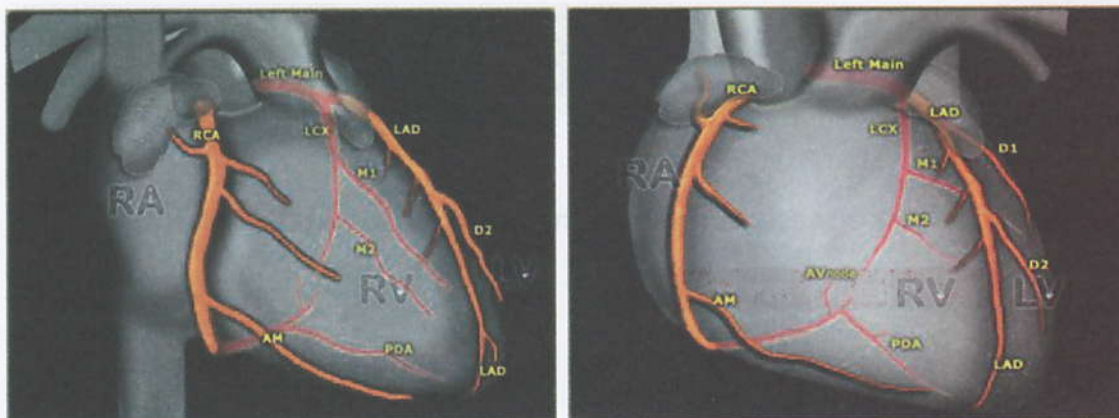
1- Left coronary artery : Passes forward & to the left in the left atrioventricular groove for a short distance & then divides into :

a) Anterior descending artery : passes downward in anterior interventricular groove to the apex & then turns backward to meet *posterior descending artery* .

b) Circumflex artery : runs posteriorly in the left atrioventricular groove to meet the *right coronary* .

2- Right coronary artery :

- It runs in right atrioventricular groove to the posterior surface of the heart to meet the *circumflex artery*.
- Posteriorly, it gives the **posterior descending artery** which runs in the posterior interventricular groove to meet the *anterior descending artery*.



Left Main or left coronary artery (LCA) :

- Left anterior descending (LAD) :
 - Diagonal branches (D1, D2)
 - Septal branches
- Circumflex (Cx) :
 - Marginal branches (M1, M2)

Right coronary artery :

- Acute marginal branch (AM)
- AV node branch
- Posterior descending artery (PDA)

Patterns of coronary supply :

Balanced circulation:

- The left coronary artery supplies :

LA , LV , anterior wall of interventricular septum.

- The right coronary artery supplies :

RA , RV , posterior wall of interventricular septum.

Left coronary predominance :

- The left coronary supplies also: posterior wall of right ventricle.

Right coronary predominance :

- The right coronary supplies also: posterior wall of left ventricle.

Presentation of ischemic heart disease :

- Asymptomatic (*silent*).
- **Angina** (Stable & unstable).
- **Myocardial infarction.**
- Heart failure, Shock.
- Arrhythmia.
- Sudden death.

ATHEROSCLEROSIS

- It is a condition in which patchy deposits of fatty material (atheromas or atherosclerotic plaques) develop in the walls of medium-sized & large arteries, leading to reduced blood flow.
- It can cause serious complications e.g.

➤ Coronary artery disease :

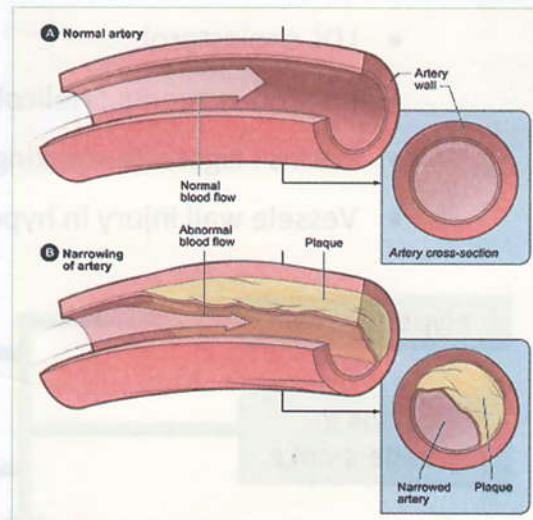
- ☠ Angina.
- ☠ MI.
- ☠ Sudden death.

➤ Cerebrovascular disease :

- ☠ Stroke.
- ☠ Transient ischemic attack (TIA).

➤ Peripheral arterial disease.

➤ Erectile dysfunction in men.



Risk factors for atherosclerosis:

Non modifiable :

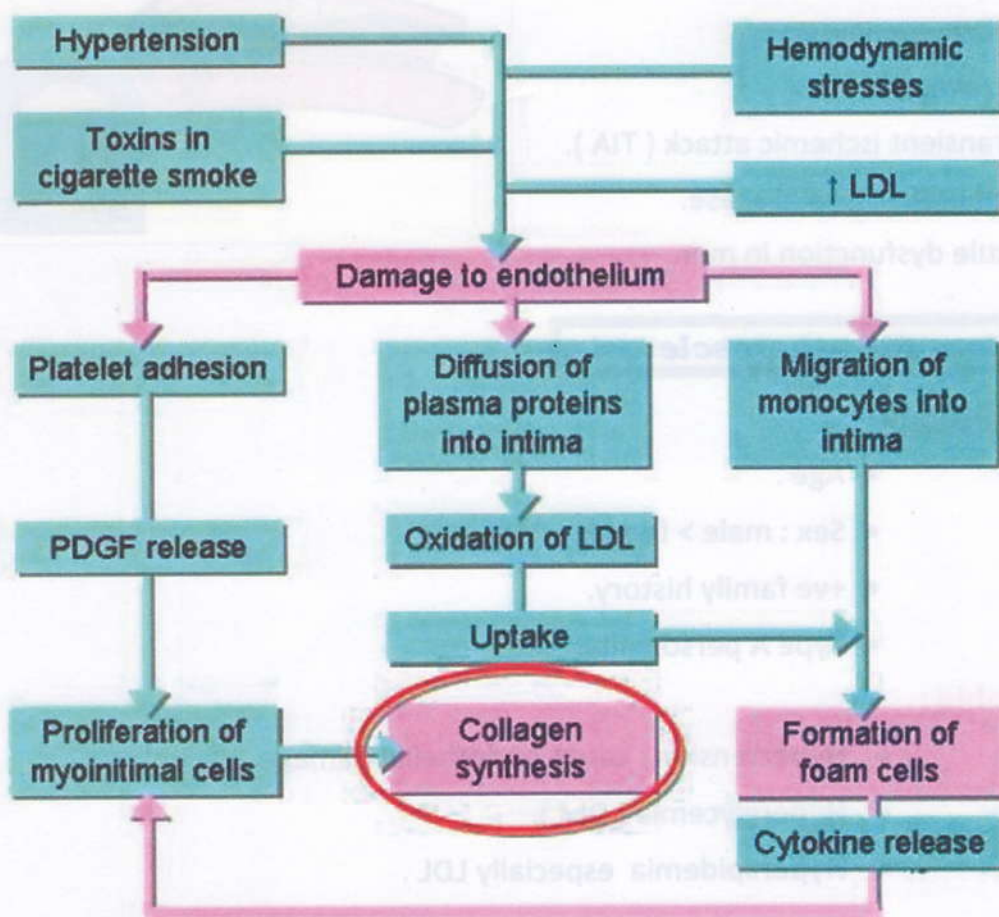
- Age .
- Sex : male > female .
- +ve family history.
- Type A personality.

Modifiable :

- **Hypertension** : cause endothelial damage.
- **Hyperglycemia** (DM).
- **Hyperlipidemia** especially LDL .
- **Hyperuricemia** .
- Sedentary life style .
- Smoking.
- Stress.

Pathophysiology of atherosclerosis :

- **" Response to injury " theory** : chronic endothelial injury → vascular inflammation & fibroproliferative response.
- **Causes of endothelial injury include :**
 - LDL cholesterol.
 - Infectious agents : Helicobacter pylori, Chlamydia pneumonia.
 - Toxins : cigarette smoking, hyperglycemia,
 - Vessel wall injury in hypertension.



STABLE ANGINA PECTORIS

Definition :

It is a **clinical syndrome** of chest pain due to imbalance between oxygen supply & demands of the myocardium.

Etiology :

I. Decreased myocardial oxygen supply :

1) Decrease in quantity :

a) Coronary artery disease :

- **Coronary atherosclerosis.** (The most common cause)
- Coronary vasculitis : polyarteritis nodosa, Kawasaki disease, SLE.
- Coronary spasm.
- Coronary embolism.
- Microvascular angina (syndrome X).
- Coronary osteial stenosis of syphilis.
- Congenital anomalies.

➤ Others :

- Amyloidosis.
- Post transplantation.
- Ionized radiation.

b) As a part of LCOP : AS , LSHF.

2) Decrease in quality :

- Anemia.
- Hypoxia.

II. Increased myocardial oxygen demand :

- Ventricular hypertrophy .
- Tachycardia .

Clinical picture :Symptoms :Chest pain with the following **7** criteria :

	Common (classic)	Less common	Never
1) Site :	• Retro-sternal Often the patient places his clenched hand over the upper sternum.	Any site of i chest : • Scapular. • Infraclavicular. • Epigastrium.	• Left infra mammary • <i>Patient never points with his finger.</i>
2) Character:	• Compressing. • Constricting.	• Heaviness. • Squeezing. • Burning. ☹ • Discomfort.	• Stitching. • Pricking. • Throbbing.
3) Radiation :	• Left shoulder & inner side of the left arm up to little finger. • Neck, jaw or teeth.	• Right shoulder . • Back. • Epigastric area.	• Below epigastrium.
4) Duration :	Less than 15 min (1 - 5 min)	More than 15 min	Never seconds or hours.

5) Precipitating factors :

- Exercise.
- Cold weather.
- Heavy meals.
- Smoking.
- Sexual intercourse
- Stress .

N.B : Many patients report a fixed threshold for angina, which occurs predictably at a certain level of activity.

6) Relieving factors :

- Rest, but occasionally the pain disappears with continued exercise (*walk through angina*)
- Sublingual nitrates.

7) Association :

- Sweating
- Dizziness
- Dyspnea : may occur due to LVF .
- Fear of death (*angor animi*)
- Eructation at the end of the attack.



Silent ischemia : Dyspnea, fatigue & dizziness with NO pain.

Signs : (during the attack) usually **NO** abnormal finding

- A positive Levine sign : characterized by the patient's fist clenched over the sternum when describing the pain.
- Pain produced by chest wall pressure is usually of chest wall origin.
- Pallor , tachycardia & hypertension (secondary to sympathetic stimulation).
- S₁ : weak .
- S₂ : reversed splitting .
- S₃ : due to LVF .
- Murmur of MR : due to papillary muscle dysfunction.
- In between the attacks :
 - Physical examination is important to exclude anemia & valvular stenosis.
 - Physical examination of abnormal lipid metabolism (e.g. xanthelasma) or of diffuse atherosclerosis (e.g. diminished peripheral pulse).

NB : I can say that the great significance of cardiac examination in a case of Angina is just for reassurance & no one can blame me !!!!!!!

Investigation :

1- ECG :

A) Resting ECG :

- **In between the attacks :**
 - usually normal.
 - ECG of old MI .
- **During the attack:**
 - ST segment : depressed. (more than 1mm)
 - T wave : Inverted .

B) Holter monitoring : to detect silent ischemia.

C) Exercise ECG : (in between the attacks only)

- The patient is exercises on a treadmill & ECG changes & vital signs are recorded.
- Stress test can be done with dobutamine in patients unable to do exertion.
- Stress test is considered +ve when : one or more of these changes are present :
 - **Symptom** : Typical anginal pain during the test.
 - **Sign** : Fall in blood pressure (10 mmHg or more) suggests ischemia
 - **ECG** : Depressed ST segment > 1mm .

NB: Exercise test can be **misleading** as there are :

- False negative test : So normal test doesn't exclude IHD .
- False positive test : especially in patients with left ventricular hypertrophy.

Stress test is contraindicated in :

- Acute attacks.
- Severe AS.
- Severe hypertension.
- Congestive heart failure.
- Orthopedic problems.

2- Echo & dobutamine Echo : may show abnormal motion of the myocardium .

3- Cardiac scan : (Radioactive Thallium 201)

Thallium 201: is taken up by healthy myocardium & not by ischemic myocardium (*cold spot*)

4- Coronary angiography : (coronary catheter)

- To detect the site & severity of coronary occlusion.
- It's generally used to determine whether mechanical revascularization (CABG or PTCA) is possible & to guide this therapy.

5- Laboratory investigations:

- For risk factors :Blood glucose level , Plasma lipid (cholesterol).
- Cardiac enzymes : normal.

Canadian Cardiovascular Society Functional Classification of Angina :

Grade	Clinical finding	Features
I	no limitation of ordinary activity	Ordinary physical activity (such as walking or climbing stairs) does not cause angina. Angina may occur with strenuous rapid or prolonged exertion at work or recreation.
II	slight limitation of ordinary activity.	Angina may occur with • walking or climbing stairs rapidly or after meals or under emotional stress.
III	marked limitation of ordinary physical activity	Angina may occur after • climbing 1 flight of stairs in normal conditions.
IV	unable to carry on any physical activity without discomfort	Angina may be present at rest.

Treatment :

4

1- Control of risk factors :

(risk factors of atherosclerosis)

- ✗ Reassurance & sedation.
- ✗ No smoking.
- ✗ Treatment of hyperlipidemia.
- ✗ Control of hypertension & diabetes.
- ✗ Change of life style (regular exercise program).

2- Medical treatment of angina :

in between the attacks

i. Nitrates :

Action :

- Venodilator → ↓ preload (venous return) → ↓ myocardial oxygen demand.
- Coronary dilatation → increase coronary blood flow. (mild effect)

Preparation :

- Nitroglycerine (*nitromack*) : 2.5 mg twice daily orally or transdermal patches.
- Isosorbide dinitrate (*dinitra*) : 10-20 mg twice daily.
- Isosorbide mononitrate (*effox*) : 20-40 mg twice daily.

Side effects :

- Headache.
- Hypotension.
- Tolerance : so start with minimal effective dose with nitrate free interval periods.

ii. β blockers :

Action :

Reduce oxygen demand since they reduce heart rate, blood pressure & contractility.

Preparation :

- Propranolol (indral) : non selective β blocker .
- Atenolol (ateno), Metoprolol (betaloc) , Bisoprolol (concor) : Selective β blockers.
- Carvedilol (cardilol) : β blocker with an arteriolar vasodilating action.

Side effects :

- Lung : Bronchospasm.
- Heart : Bradycardia , Heart block.
- Depression , Impotence.

iii. Calcium channel blockers :

Action :

- Reduce oxygen demand by : $\downarrow$ -ve inotropic action.
 $\downarrow$ afterload (arteriolar dilators).
- Coronary dilator : increase coronary blood flow (*effective in variant angina*)

Preparation :

- Verapamil (*Isopten*) : great -ve inotropic & weak vasodilator: 80 mg t.d.s.
- Diltiazem : 60 mg twice daily.
- Nifedipine (*adalat*) : mainly vasodilator & weak -ve inotropic: 10 – 20 mg t.d.s.
- Recently : **Amlodipine** (*norvasc*) : mainly vasodilator.

Side effects :

- Headache.
- Hypotension.
- Precipitation of Heart failure.
- Constipation.
- Peripheral edema. MCQ
- Verapamil & Diltiazem : bradycardia & heart block.

iv. **Antiplatelet :**

- Aspirin : 75 mg single dose : it improves the prognosis.
- Clopidogrel (*plavix*) : expensive.

3- Coronary revascularization :

Indications :

- Angina not responding to medical treatment.
- Post infarction angina to improve the prognosis.

Techniques :

PTCA (Percutaneous Transluminal Coronary Angioplasty):

Introduction of balloon or *stent* to dilate the stenotic artery(*balloon-tipped catheter*)

Indication of PTCA :

Stenosis of one or two vessels only (except left main coronary artery)

CABG (Coronary Artery Bypass Graft) :

Grafting a piece of saphenous vein or internal mammary artery between the aorta & the coronary artery distal to any obstruction.

Indication of CABG :

- Stenosis of 3 or more vessels.
- Stenosis of left main coronary artery.
- For diabetic patients with 2 or 3 - vessel disease.

4- Treatment of anginal attack :

- Complete rest.
- Nitroglycerine (0.5 mg) or isosorbide dinitrate (5mg) sublingually & repeated up to 3 times successively with interval of 3 minutes.

NB : If the patient is not relieved after the use of 2-3 tablets ,the patient should be immediately transferred to hospital & evaluated for the possibility of myocardial infarction.

ACUTE CORONARY SYNDROME

1. Unstable angina.
2. ST elevation myocardial infarction (STEMI).
3. Non ST elevation myocardial infarction (NSTEMI).

UNSTABLE ANGINA

– Definition :

- Change in the character of angina : ↑ frequency, severity or duration.
- It is considered intermediate syndrome between stable angina & MI.

– Etiology :

- Non occlusive coronary thrombosis on top of atherosclerosis.
- Post MI angina.
- Recent onset angina.
- **Coronary artery spasm** : Prinzmetal (*Variant*) angina.
 - Caused by **spasm** of coronary artery with or without atherosclerosis.
 - Unpredictable, at rest.
 - ECG : Transient ST **elevation**.
 - Treatment :
 - β blockers are **contraindicated** (↑ *coronary spasm*) . ☒
 - Nitrate & Ca Channel blockers are drugs of choice.

– Investigations of unstable angina :

- ECG & coronary angiography.
- Cardiac enzymes : are **NOT** elevated ,to differentiate it from MI.

- Treatment : to reduce progression to MI

I. Medical :

- ✎ IV infusion nitroglycerin : 5-10 μ g/min and is raised gradually.
- ✎ β blockers.
- ✎ Anti-platelets : Aspirin, Clopidogrel (*Plavix*)
- ✎ LMW heparin : enoxaparine (*clexane*)

II. Coronary revascularization : PTCA or CABG.

Decubitus Angina : usually on lying down (occurs in HF).

Nocturnal Angina : It awakens the patient from sleep , associated with dreaming .

Angina of Lewis : in cases of AR , it is nocturnal & prolonged.

Acute Myocardial Infarction

Definition :

Ischemic necrosis of part of the cardiac muscle due to sudden, persistent & complete cessation of its blood supply.

Etiology :

- **Thrombosis on top of atherosclerosis.** 🎵🎵
- **Non-atherosclerotic causes of myocardial infarction :**
 - Coronary artery diseases : spasm, dissection, PAN, Takayasu's disease.
 - Coronary angiography.
 - Aortic stenosis, regurge & prolonged hypotension.
 - Polycythemia, Sickle cell anemia, DIC, TTP.
 - Embolism : IE, Artificial valve, myxoma.

Pathology :

➤ Gross pathology : ○ No changes for 8-12 h ○ Then area become dark. ○ After 24 h pale centre, normal edges.	➤ Microscopic appearance : ○ Coagulative necrosis of myocardial tissue. ○ Cellular infiltration and inflammatory response. ○ Fibroblast appear & replacement with fibrous tissue.
--	---

Classification :

Site:

- 1- Occlusion of the left anterior descending artery ➔ anterior infarction.
- 2- Occlusion of the circumflex artery ➔ lateral infarction.
- 3- Occlusion of the right coronary artery ➔ inferior infarction.

Types :

- **Transmural infarction** (**ST** elevation myocardial infarction - **STEMI**) : infarction of full thickness of the ventricular wall.
- **Subendocardial infarction** (**Non ST** elevation myocardial infarction - **NSTEMI**) :
Transient or incomplete vessel occlusion.

Clinical picture :**Pain and/or complications****I. Chest pain:** Similar to **angina** but :

- More severe, it may be severe enough to be described as the worst pain the patient has ever felt.
- Radiates more : may below epigastric area but never below umbilicus.
- More prolonged : up to several hours.
- Unrelated to precipitating factors : may at rest.
- Not relieved by rest or sublingual nitrate.
- Associations: like angina & may also associated with complications.

NB: Painless infarction:

- Elderly.
- Diabetic neuropathy.
- Patient under anesthesia.
- Transplanted heart (denervated).

II. Complications :**6 early & 6 late****Early complications :****6 items****1- Shock :** see p139

<i>Cardiogenic shock</i>	<i>Neurogenic shock</i>
Caused by massive infarction (> 40% of the cardiac muscle) leading to severe pump failure & high jugular venous pressure.	Caused by severe pain (vagal stimulation).
C/P : Hypotension , tachycardia ,pulmonary edema.	C/P : Hypotension, bradycardia .
ttt: The same as acute pulmonary edema & mechanical assist devices: intra-aortic balloon counterpulsation.	ttt : morphine .
Prognosis : very bad.	Prognosis : good .

2- Acute heart failure : with normal heart size.

3- Arrhythmia :

- All types may occur.
- The most serious are : VT , CHB .

4- Myocardial rupture :

- Rupture of the septum → acquired VSD .
- Rupture of papillary muscles → acute MR → acute heart failure.
- Rupture of the ventricular free wall → blood fills the pericardium → cardiac tamponade.

5- Dry pericarditis : Hemorrhagic pericardial effusion may develop especially with thrombolytic therapy.

6- Sudden death :

- Arrhythmia (VT , VF) : most deaths occur during few hours after MI .
- Acute heart failure.
- Cardiogenic shock.
- Cardiac rupture.

Late complications :

6 items

1- Post infarction syndrome : (*Dressler's syndrome*) within 4 weeks or more

Autoimmune phenomenon in response to necrotic cardiac tissue characterized by :

- Pericarditis
- Pleurisy
- Pneumonitis
- fever.

2- Post infarction angina :

Due to affection of other diseased coronaries.

3- Myocardial aneurysm : (*dilatation of the scar tissue of MI*)

- On examination : double apex .
- ECG : persistent ST segment elevation .
- Fate :
 - Refractory heart failure.
 - Rupture aneurysm.
 - Recurrent embolism.
 - Recurrent arrhythmia.

4- Thrombo-embolism :

- **Mural thrombosis :** (infarction → rough surface → thrombosis → systemic emboli)
- **DVT :** due to prolonged recumbency → pulmonary embolism .

5- Frozen shoulder : stiffness with limitation of movement due to :

Pain → reflex arteriolar spasm & ischemia.

→ may be psychic.

6- Complications of treatment: anticoagulant , prolonged bed rest,....**Signs :**

(not specific)

nothing or anything

- The physical examination may be entirely normal.
- Pallor , sweating , nausea , vomiting & fever.
- **Pulse :**
 - Tachycardia : sympathetic stimulation , cardiogenic shock .
 - Bradycardia : neurogenic shock , HB , inferior MI.
 - Irregular : arrhythmias.
 - weak : LVF .

NB : Bradycardia is often seen with inferior MI because the right coronary artery supplies the SA node.

- **Blood pressure :**
 - Hypertension : sympathetic stimulation .
 - Hypotension : LVF , shock .
- **Cardiac auscultation :**
 - S1 : weak.
 - S2 : reversed splitting.
 - S3 : due to LVF.
 - S4 : due to decreased myocardial compliance.
 - Murmur : of MR , VSD .
 - Pericardial rub : Dry pericarditis.
- **Congested neck vein :** in right ventricular infarction.

Differential Diagnosis :

Causes of acute chest pain :

- Stable angina.
- Unstable angina.
- MI.
- Pulmonary embolism.
- **Aortic dissection.** ☠
- Pneumothorax.
- **Acute dry pericarditis.** ☠
- Cardiac neurosis.
- Esophageal spasm , Perforating peptic ulcer , Cholecystitis.

Investigations:

1- Cardiac enzymes :

Cardiac enzymes are released into blood from necrotic heart muscle after an acute MI.

Marker	Initial rise	Return to normal	Notes
Creatine phosphokinase (CPK)	4-8 h	2-4 days	Non specific because it may rise in damaged skeletal muscles or brain.
CPK-MB	4-8 h	2-4 days	It's isoenzyme of CPK , specific to cardiac muscle
Lactic dehydrogenase (LDH)	10 h	1-2 weeks	Not specific .
Troponin (cTnT , cTnI)	3-12 h	1 week	<u>Most sensitive & specific</u> markers of myocardial damage .
Myoglobin	1-4 h	24 h	

2- ECG :**➤ In transmural infarction (ST Elevation MI):**

1. Convex **elevation** of ST segment.

2. T wave :

- Tall (hyperacute) in the first few minutes after vessel occlusion (*the earliest change*)
- later on : **Inverted** T wave (representing severe ischemia)

3. Finally, **pathological Q waves** occur, representing significant myocardial necrosis & replacement by scar tissue.

(Pathologic Q waves are usually defined as duration ≥ 0.04 s or $>25\%$ of R-wave amplitude)

➤ In subendocardial infarction (Non ST Elevation MI) :

1. ST segment : normal or depressed.
2. No pathological Q waves (non Q wave MI)
3. T wave : inverted.

NB: The ECG may be normal during the first few hours of infarction .

In old MI : The only residual change is the pathological Q wave.

3- Echocardiography :

- Ventricular wall motion abnormalities.
- Complications : MR , myocardial aneurysm.

4- Cardiac scan : Like angina .**5- Coronary angiography :**

reveals which vessels have been affected and the extent of damage.

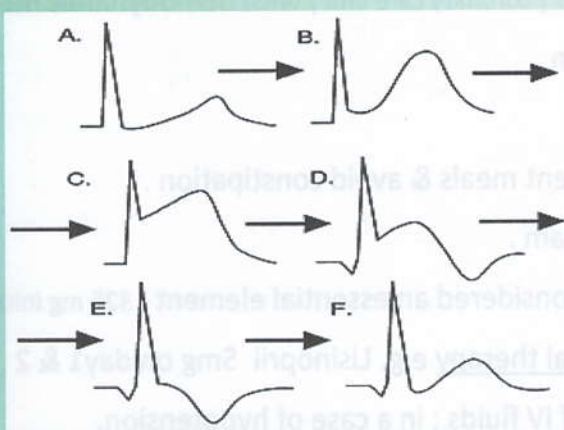
6- Leukocytosis , $\uparrow$ ESR : as there is tissue damage.

Site of infarction as regard to ECG

localization	ST elevation	coronary artery
Anterior MI	V1-V6	LAD
<u>L</u> ateral MI	I, aVL, V5, V6	RCX or MO
In <u>f</u> erior MI	II, III, aVF	RCA (80%) or RCX (20%)

**ECG evolution of STEMI**

Not all of the following patterns may be seen; the time from onset of MI to the final pattern is variable and related to the size of MI, the rapidity of reperfusion, and the location of the MI.



- A. Normal ECG prior to MI
- B. Hyperacute T wave changes - increased T wave amplitude and width.
- C. Marked ST elevation with hyperacute T wave changes (transmural injury)
- D. Pathologic Q waves, less ST elevation, terminal T wave inversion (necrosis)
- E. Pathologic Q waves, T wave inversion (necrosis and fibrosis)
- F. Pathologic Q waves, upright T waves (fibrosis).

Diagnosis of MI :

At least 2 of the following 3 criteria :

1. Classic chest pain.
2. ECG changes.
3. Positive biomarkers (cardiac enzymes)

Treatment :3 X **I. Pre hospital :**

- 1- Rapid transfer to hospital is a must (*Time lost is lives lost*) .
- 2- Oxygen inhalation.
- 3- Analgesics for pain → Morphine 5 - 10 mg IV
- 4- For ventricular arrhythmias → Lidocaine 50 – 100 mg IV ??
- 5- For heart block → Atropine 0.5 – 1 mg IV .

II. Hospital care : **1- General :**

- a. Admission to CCU (coronary care unit) with hemodynamic monitoring & continuous ECG
- b. Oxygen inhalation .
- c. Complete rest .
- d. Diet : Light frequent meals & avoid constipation .
- e. Sedative : Diazepam .
- f. Aspirin : is now considered an essential element (325 mg initial dose then 75 mg daily-oral)
- g. ACE Inhibitor: Oral therapy e.g. Lisinopril 5mg on day1 & 2 ,then 10 mg daily.
- h. Administration of IV fluids : in a case of hypotension.

NB : ACE Inhibitors are vasodilator that reduce cardiac work & decrease myocardial energy requirement .

ACE Inhibitors also have inhibitory effect on the cardiac remodeling.

2- Relieving of chest pain :

- a. Morphine (4 mg IV every 5 to 10 minutes as needed)
 - b. Nitroglycerine .
 - c. β blockers .
- } to relieve pain of post infarction angina.

3- Thrombolytic therapy : (*time is muscle*)

- The earlier that thrombolytic therapy is given after the onset of chest pain, the greater the benefit (*thrombolytic therapy is beneficial up to 6 hours but may be given for up to 12 hours*)

Drugs :

- ✎ Streptokinase : 1.5 million units IV over 60 min. may cause allergy .
- ✎ Urokinase.
- ✎ Alteplase (tissue plasminogen activator – tPA)

The important issue in thrombolytic therapy is not which drug to use, but how quickly to use it.

- Anticoagulant (*heparin*) & antiplatelet (*aspirin*) are given with & after thrombolytic therapy to reduce the risk of reocclusion.

Contraindications : the major risk is **Bleeding**

- Bleeding disorders.
- Major surgery within past 2 weeks .
- Recent cerebral hemorrhage within past 12 months.
- Active internal bleeding e.g. peptic ulcer.
- Sever hypertension.
- Diabetic retinopathy with recent bleed.
- **Aortic dissection.**
- Pericarditis.

4- Angioplasty : Percutaneous Transluminal Coronary Angioplasty (PTCA) :

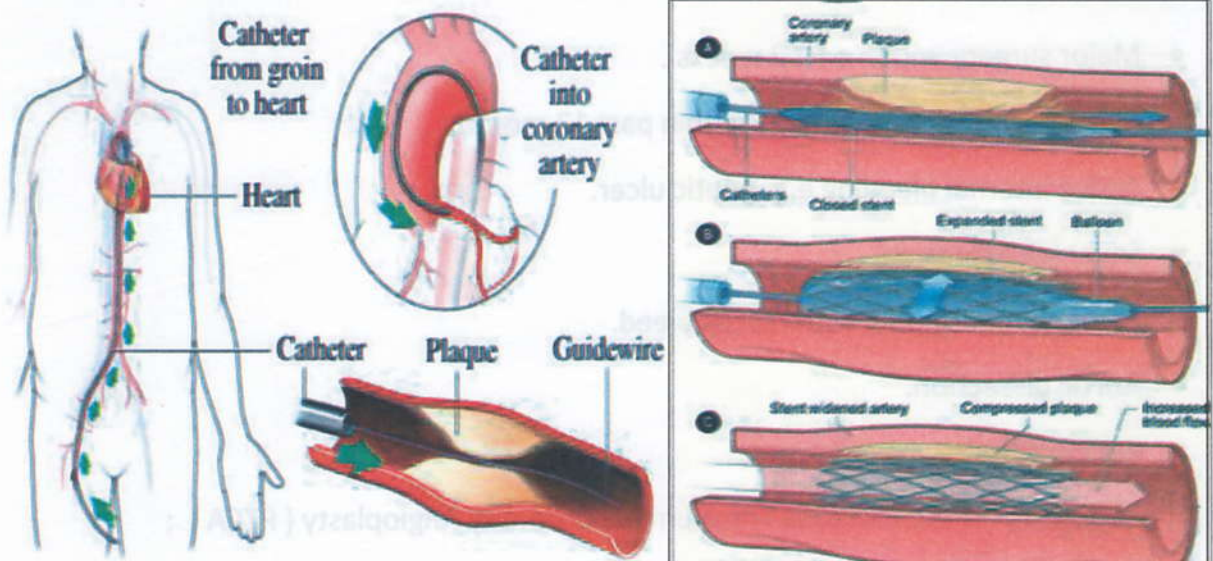
- Introduction of balloon or *stent* to dilate the stenotic artery (*balloon-tipped catheter*)
- More effective than thrombolytic therapy (fewer complication , shorter hospitalization).

5- Treatment of early complications : e.g.

- Acute heart failure.
- Arrhythmia.
- Cardiogenic shock.

III. After discharge : (ABCDE)

1. A : Aspirin, ACEIs.
2. B : B blockers, BP control.
3. C : Cholesterol control.
4. D : Diabetes control, diet.
5. E : Education, reassurance & rehabilitation.



Percutaneous Transluminal Coronary Angioplasty
(PTCA)

6

Rheumatic Fever

Definition:

Diffuse inflammatory disease typically caused by an abnormal immune response to upper respiratory tract infection with group A - β hemolytic streptococci.

Etiology :

There are 3 theories

- Delayed immune response to pharyngeal infection with rheumatogenic strains of group A beta hemolytic streptococci.
- After a latent period of 1-3 weeks, antibody induced immunological damage occur to heart valves, joints, SC tissue, basal ganglia

1- Antigenic similarity : (Cross reactivity theory)

The antigen of streptococci is similar to the antigens of heart & connective tissue So, antibodies produced against streptococci can react with cardiac muscle & other CT ..

2- Altered antigenicity : (Autoimmune theory)

Streptococci or its toxins may change the character of the connective tissue making it abnormal to the body so acting as an antigen $\rightarrow$ $\uparrow$ of auto antibodies against it.

3- Hypersensitivity theory :

Streptococcal antigens $\rightarrow$ $\uparrow$ antibodies + complements $\rightarrow$ complex mediated injury.

Pathology :

1- Site : (*Rheumatic fever bites the heart & licks the joints*)

- 1- Joints : synovial membrane ..
- 2- Heart : Pancarditis ..
- 3- CNS ..
- 4- Skin & subcutaneous tissue ..
- 5- Small blood vessels ..
- 6- Serous membrane .. (*Pleura , Peritoneum , Pericardium*)

II- Types : *There are 2 types***1- Exudative lesion :**

- Affecting mainly the serous membrane .
- Heals mostly without scarring or deformity .

2- Proliferative lesion : formation of Aschoff nodules

- Affects mainly the heart & the skin.
- Heals by fibrosis .

➤ Consists of : (from in to out)

- ✓ Fibrinoid degeneration .
- ✓ Lymphocytes & plasma cells .
- ✓ Aschoff giant cells .
- ✓ Fibroblasts .
- ✓ Layer of fibrosis .

Predisposing factors :

- 1- **Recurrent streptococcal infections** of the pharynx & tonsils .
- 2- Age : most common between 5-15 years (rare below 3 & above 30 y) .
- 3- Sex : equal in both , but chorea is more common in females.
- 4- Familial : due to similar environment (e.g. overcrowding) & genetic predisposition.
- 5- Common in 3rd world countries.

Clinical picture :**Latent period :** 1- 3 weeks**I- Major criteria :** (mnemonic : **C.A.N.C.E.R**)**1- Carditis :** found in 50 % of patients

Rheumatic fever affects the 3 layers of the heart (pancarditis)

➤ Pericarditis :

- Dry pericarditis : causing stitching pain & pericardial rub .
- Pericardial effusion : less common .
- Never constrictive pericarditis .

➤ **Myocarditis :**

- Tachycardia : not proportional to the degree of fever.
- Tic - Tac rhythm : due to loss of muscular component of S1 .
- Heart failure .
- Arrhythmia & may be heart block .

➤ **Endocarditis :** (valvulitis)

- Order of frequency : **Mitral** > **Aorta** > **Tricuspid** > **Pulmonary** (*MAT P*)
- Carey Coomb's murmur: Transient mid diastolic murmur of mitral stenosis in acute stage due to swelling of the mitral cusps causing narrowing of the mitral valve .
- Later on (over years): fibrosis may lead to stenosis , regurge or both .

2- **Arthritis :** found in 75% of patients

- Polyarthritis .
- Affects big joints : Knees , ankles , elbows .
- Asymmetrical .
- Migratory (flitting)
- The affected joints show hotness , redness , tenderness , swelling with limitation of movement .
- Dramatic response to salicylates .
- Duration of arthritis is about 2-6 weeks .
- No deformity : leaving the joints with complete resolution .

👍 In **Children** : Carditis > arthritis .

👍 In **Adult** : **Arthritis** > carditis .

3- **Subcutaneous Nodule :** rare

- Description: Small ,painless ,tenderless ,firm nodule ¬ adherent to skin.
- Etiology : accumulation of Aschoff nodule .
- Site : over bony prominences & tendons e.g. dorsum of the hands & feet
- Importance : associated with severe carditis .

4- **Chorea** : (Sydenham's chorea) : 10 % of cases

- Involuntary , sudden movements with emotional instability... See neurology
- More common in female.
- It appears very late after streptococcal infection (6 months), so it's not associated with arthritis & ESR is not raised.
- It is self limiting.

5- **ERYthema marginatum** :

- Occurs in the form of areas of erythema with central pallor.
- Site : trunk & proximal part of limbs.
- Non pruritic , non painful.

II- **Minor criteria** : 🏆 (mnemonic : P.E.A.C.E)

1. **P**rolonged PR interval in the ECG : > 0.22 sec , due to myocarditis .
2. **E**SR : elevated .
3. **A**rthralgia : painful joints without swelling.
4. **C**RP (C reactive protein) : elevated .
5. **E**levated temperature (fever) .

III - **Other manifestations of rheumatic fever** :

- Pallor .
- Pleurisy.
- Pneumonia.
- Peritonitis ⇒ Abdominal pain.

Diagnosis of rheumatic fever : (Duckett Jones criteria)

- ✓ 2 major criteria or 1 major & 2 minor criteria. *plus,*
- ✓ Evidence of recent streptococcal infection (+ve throat culture , ↑ASO , Scarlet fever)

- No need to say that if arthritis is taken as a major criteria , don't consider arthralgia as a minor criteria.
 - Also if carditis is taken as a major criteria , don't consider prolonged PR interval as a minor criteria.

Complications :

- ↳ **Acute complications :** i. Heart failure ii. Arrhythmia & heart block.
- ↳ **Chronic complications** = Complications of valvular heart diseases .

Differential diagnosis :

- Fever of unknown origin.
- Infective endocarditis.
- Juvenile rheumatoid arthritis & bacterial arthritis.
- Systemic lupus erythematosus (SLE).
- Acute leukemia.
- Henoch-Schonlein purpura.

Investigations : There is NO specific confirmatory test .**a) Laboratory :** (evidence of recent streptococcal infection)

- Blood picture : Leucocytosis.
- ESR : ↑↑
- CRP (C reactive protein) : ↑↑
- ASO titre (*Anti Streptolysin O titre*) : Normally up to 150 Todd units
Higher than 250 Todd units in adult & 333 Todd units in children indicates recent streptococcal infection .
- Throat culture : may reveal group A streptococci .

b) Cardiac : (evidence of carditis)

- X ray : Cardiac enlargement.
- ECG : Prolonged PR interval , arrhythmia & heart block.
- Echo : Cardiac enlargement & valve lesions.

Signs of rheumatic activity :

- ↳ Major or minor criteria.
- ↳ Laboratory tests: ↑ ESR , CRP , ASO & Leucocytosis.

Treatment :**Prophylactic :****i. Primary prevention :** (to prevent rheumatic fever)

- ✗ Proper management of upper respiratory tract infection.
- ✗ Tonsillectomy of chronically infected tonsils.

ii. Secondary prevention : (to prevent recurrence of rheumatic activity)

- ✗ Long acting penicillin: Benzathine penicillin (*Retarpen*): 1.2 million U/2-4 weeks **IM**
Or
- ✗ Penicillin V (*Ospan*) 250 mg /12 hr Orally . or
- ✗ Erythromycin 250 mg /12 hr Orally . (in a case of allergy to penicillin)

For 5 years after the last attack or till age of 25 (which is longer) & may for ever.

iii. Tertiary prevention : prevention of infective endocarditis.**Curative :**

1- Complete bed rest : till improvement of all symptoms & signs.

2- Diet : Light meals & salt restriction.

3- Antibiotics :

- ✗ Benzyl penicillin G (*Crystalline penicillin*) : One IM injection (600.000 to 1.200.000 units) for about 10 days.
- ✗ Penicillin V (*Ospan*) : 250 mg / 6 hr orally for about 10 days.
- ✗ Erythromycin 250 mg / 6 hr orally for about 10 days. (in a case of allergy to penicillin)

4- Anti-inflammatory drugs :

✗ **Aspirin :** for **Arthritis**

- 100 mg / kg / day in 6 divided doses up to satisfactory laboratory findings .
- Gradual withdrawal is important to avoid rebound arthritis.
- S/E of aspirin : *mnemonic* ASPRIN ... see Rheumatology book P 14

✎ **Corticosteroids :** for **C**arditis ?

- Predinsone (*Hostacortin*) : 1 mg / kg / day in 4 divided doses for 4 weeks then gradual withdrawal in 4 weeks .
- Aspirin should be added during steroid withdrawal to avoid rebound activity.

5- Treatment of complications :

➤ Congestive heart failure :

- Digitalis , Dilator , Diuretic.
- Unlike normal heart failure, rheumatic heart failure responds well to cortisone.

➤ Chorea :

- ✎ Tranquilizer : Chlorpromazine.
- ✎ Haloperidol : is effective in severe cases.

Try to learn something about everything and everything about something.

Thomas Henry Huxley

7

Infective endocarditis

Definition : Infection of the endocardium.

Classification :

There are 2 forms depending on the virulence of the organism :

1. **Subacute IE** : most common & requires combinations of the 2 factors (infection & cardiac lesion e.g. damaged valves).
2. **Acute IE** : affecting the healthy endocardium (normal valves), usually occurs in the right side of the heart in addicts. Caused by Staphylococcus aureus.

Etiology :

The development of infective endocarditis requires combination of 2 factors :

- I. Infection.
- II. Underlying cardiac disease.

I- Infection : (any organism but commonly bacteria)

a) **Gram +ve cocci** :

- o **Strept. viridans** (the most common organism): e.g. tooth extraction.
- o Strept. faecalis : e.g. GIT procedures.
- o Staph. aureus :e.g. cardiac catheterization.
- o Staph. epidermidis : accounts for 30% of cases of prosthetic valve endocarditis.

b) **Gram -ve bacilli** : Haemophilus influenza , Klebsiella, Pseudomonas.

c) **Others** : Fungi , Rickettsia , Chlamydia

II- Underlying cardiac disease :

(In fact, all situations inducing a turbulent blood flow are a favorable to an infective endocarditis)

a) **Valvular disease** : (left > right , regurge > stenosis)

- o All valvular heart diseases except MS (due to marked fibrosis) .
- o Prosthetic (artificial) valve endocarditis.

b) Congenital heart disease :

- Especially : small VSD , PDA, coarctation of aorta, pulmonary stenosis.
- Uncommon in ASD (low pressure gradient between the 2 atria).

c) Myocardial infarction.

Pathology :

- **Vegetations** are formed over the valvular & mural endocardium causing valvular damage. (The organism in the center surrounded by fibrin & platelets) → Vegetation may detach leading to embolization.
- Left sided in 90% of cases, rare on the right side.
- Notice that this vegetation is big , friable & septic.
- Also the myocardium may contain foci of inflammation.

Clinical picture :

Manifestations of the disease are due to :

- Toxemia.
- Embolization.
- Immune complexes.

A) Extra cardiac manifestations :

Face :

i. Fever (usually low grade & prolonged).

Any prolonged unexplained fever in cardiac patient is considered & treated as infective endocarditis until proved otherwise.

ii. Pallor & toxic facies.

iii. Eye :

- ✓ Subconjunctival hemorrhage . (Toxemia)
- ✓ Roth spots (area of retinal hemorrhage with pale center)
- ✓ Sudden blindness : due to embolism of the central retinal artery.

Upper & lower limbs :

- i. **Clubbing** : It occurs with long standing cases .
- ii. **Splinter hemorrhage** : longitudinal hemorrhage under the nails due to rupture capillaries (toxemia)
- iii. **Osler's nodule** : Small , painful , intracutaneous in the pulps of fingers & toes.(due to toxic endothelial hyperplasia of the capillary .
- iv. **Janeway's nodule** : Small painless patches on the palms .
- v. **Abnormalities of the radial pulse** :
 - ✓ **Tachycardia** : due to fever .
 - ✓ **Absent pulse** : due to embolization of brachial artery.

Spleen :

- **Infection** : mild enlarged tender spleen in 80 % of cases.
- **Infarction** : Stitching pain & splenic rub.

Kidney :

- **Infection (Immune complexes)** : It presents clinically as nephritic syndrome, nephrotic syndrome, up to chronic renal failure.
- **Infarction** : loin pain & hematuria.

CNS :

- **Infection** : Meningitis, encephalitis & mycotic aneurysm.
- **Infarction** : Embolic hemiplegia , Subarachnoid hemorrhage.

Lung :

- **Infection** : pneumonia.
- **Infarction** : due to pulmonary embolism in right sided endocarditis .(rare)

B) Cardiac manifestations :

- 1- Features of the underlying cardiac disease which already existed before IE.
- 2- Precipitation of heart failure : due to
 - Increased valvular damage.
 - Toxic myocarditis.
- 3- New murmurs due to perforated cusps or rupture of chordae tendineae.

Investigations :**1- Blood culture :** for organism (*the most important investigation*)

- ✓ It is +ve in most cases (90 %)
- ✓ At least 3 samples are taken during fever & cultured under aerobic & anaerobic conditions.
- ✓ The result are observed from 3 days up to 3 weeks.
- ✓ **Negative culture may be seen in :**
 - Antibiotic treatment before taking the blood sample .
 - Some organisms e.g. fungi .

2- Echocardiography : for vegetations

- Small vegetations can detected by trans esophageal echo.
- This is an important investigation for the diagnosis & monitoring the disease.

3- Blood picture :

- Anemia .
- Leucocytosis.
- ↑ ESR .

4- Urine analysis : for proteinuria & hematuria .**5- Immune complexes :** are presented in the circulation of most patients .

Duke's criteria for the diagnosis of infective endocarditis :**Major criteria :****1) Positive blood culture for infective endocarditis :**

Typical microorganism consistent with IE from 2 separate blood cultures, as noted below :

- viridans streptococci, Streptococcus bovis. or
- community-acquired Staphylococcus aureus or enterococci, in the absence of a primary focus.

2) Echocardiogram finding :

- ✓ Intracardiac mass on valve or supporting structure.
- ✓ Abscess.
- ✓ New valve regurgitation.

Minor criteria :

- 1) Fever > 38
- 2) Echocardiogram : finding may be consistent with IE but not the classic.
- 3) Immunological : Roth spots , Osler nodes , GIN , Rheumatoid factor.
- 4) Microbiological evidence: positive blood culture but does not meet a major criterion.
- 5) Serological studies support an infection.
- 6) Vascular : major criteria emboli , mycotic aneurysm, conjunctival hemorrhage, Janeway lesions.

Clinical criteria for infective endocarditis requires:

- Two major criteria, or
- One major and three minor criteria, or
- Five minor criteria.

Infective endocarditis should be differentiating from rheumatic fever :

	RHEUMATIC FEVER	INFECTIVE ENDOCARDITIS
Fleeting arthritis	Yes	No
Erythema marginatum	Yes	No
Petechiae	No	Yes
Hematuria	No	Yes
Clubbing	No	Yes
Splenic enlargement	No	Yes
Embolization	No	Yes
Culture	-ve	May be +ve

Treatment :**I- Prophylactic :**

a) Correction of the underlying cardiac lesion e.g. closure of VSD.

b) Prevention of infection : *Antibiotics*

➤ For tooth extraction & upper respiratory tract surgery :

Amoxicillin 2g orally 2 hours before & 2g orally 6 hours after.

➤ For GIT & genitourinary procedures :

Ampicillin : 2 g IV plus Gentamycin : 80 mg IV 2 hours before & 6 hours after.

➤ For cardiac surgery & catheterization :

Cefotaxime : 2g IV , 2 hours before & 1 gm / 6h for 1 day after.

II- Curative :**a) General :**

- Bed rest with excess vitamins.
- Hospital or CCU admission according to clinical picture.

b) Medical treatment :

1- Antibiotics :

- Once infective endocarditis is suspected , the patient must rest in bed & blood samples are taken for culture & sensitivity test .
- Treatment with antibiotics must start immediately without waiting for the result of blood culture.
- Strong antibiotics in large doses are given parenterally for at least 4-6 weeks.
- Start with : (*even before the result of blood culture*)
 - ✎ **Penicillin G** : 24 million units/day IV. **plus**
 - ✎ **Gentamycin** 80 mg /8h IM.
- **Resistant cases** should be treated according to the result of blood culture e.g. :
 - ✎ For **Strept. viridans** : penicillin G + gentamycin as before
 - ✎ For **Staphylococci** : ox , clox , dicloxacillin . or vancomycin 1gm/12h IV.
 - ✎ For **pseudomonas** : ceftazidime 2g /8h or carbinicillin .
 - ✎ For **fungal infections** : Amphotericin B .

2- Treatment of complications : e.g. heart failure.

NB : Heparin & other anticoagulants are contraindicated in IE for fear of rupture of mycotic aneurysm .

c) Surgical treatment :

- Replacement of the severely damaged valve.
- Replacement of infected prosthetic valve.
- Removal of myocardial abscess.

8

Pericardial diseases

1- Acute dry pericarditis

Etiology :

- 1- Idiopathic : most probably viral.
- 2- Infection : ◇ Viral : Coxsackie B or Echo virus (commonest) ◇ Bacterial : TB.
- 3- Infarction : may occur as early or late complication.
- 4- Irradiation .
- 5- Immunological : SLE , RA .
- 6- Iatrogenic : hydralazine , procainamide .
- 7- Infiltration : leukemia , lymphoma , breast cancer .
- 8- Rheumatic fever .
- 9- Renal failure .
- 10- Trauma : post cardiectomy syndrome .

Clinical picture :

- 1- General symptoms : FHMA (fever, headache , malaise , anorexia)
- 2- Local symptoms : **Pain**.
- 3- Signs : Pericardial rub.
- 4- Features of the cause.

The characteristics of the pain :

- **Site** : pericardial.
- **Character** : severe stitching.
- **Radiation** : may radiate to the shoulders.
- **Duration** : continuous.
- **Increased by** : movement & respiration.
- **Decreased by** : sitting & leaning forward.
- **Associated with** : pleurisy.

The characteristics of the pericardial rub :

- It's due to friction between the 2 layers of the inflamed pericardium .
- Character : superficial lathery sound .
- Timing : not related to the cardiac cycle .
- Increased by : pressing the stethoscope against the chest wall .
- Decreased by : development of effusion .
- NB : This sound not disappear with holding the respiration.

DD between acute myocardial infarction & acute pericarditis :

	MI	Pericarditis
Chest pain	Rarely affected by respiration or movement	Worse with breathing and movement.
Radiation	Often arms & jaw	Rarely arm or jaw
Fever	2-3 days after onset	At onset.
Pericardial rub	Transient	Persistent

Investigations:

ECG : Elevated ST segment & inverted T wave.

DD of elevated ST segment

Acute pericarditis	Myocardial infarction	Prinzmetal angina
Concave elevation	convex	flat
In all leads	In some leads	In some leads
Pathological Q :absent	May present	absent
Cardiac enzymes : normal	elevated	normal

Treatment :

- Treatment of the cause
- Analgesics & Anti inflammatory : Indomethacin .

2- Pericardial effusion

Etiology :

according to the fluid in the pericardial sac

Exudate (seropericardium) : $\uparrow$ cell , $\uparrow$ protein content $> 3\text{gm\%}$ & $\uparrow$ specific gravity > 1018

- It's due to $\uparrow$ capillary permeability .
- Produced by all causes of dry pericarditis .
- TB is the most common cause of pericardial effusion.
- Hemorrhagic effusion : exudate with excessive RBCs e.g. malignancy , MI , TB , CRF.

Transudate (hydropericardium) : low cell & protein content $< 3\text{gm\%}$ & $\downarrow$ specific gravity < 1018

The same causes of generalized edema :

- Heart failure $\rightarrow \uparrow$ hydrostatic pressure .
- Liver cirrhosis, Nephrotic syndrome , Malnutrition $\rightarrow$ hypoproteinemia.

Hemopericardium : Accumulation of blood

- ◇ Trauma .
- ◇ Rupture aortic aneurysm .

Chylous effusion : Accumulation of lymph e.g. Obstruction or injury of thoracic duct .

Hemodynamics :

- It depends on the amount & rate of accumulation .
e.g. 200 ml with rapid rate of accumulation may lead to cardiac tamponade & in contrast a slowly developing effusion of 2 litres can be accommodated by pericardial stretching .
- The effusion results in compression of the heart so interferes with cardiac relaxation & limit ventricular filling .
- It affects right side of the heart more than left side due to high pressure in the left side.
- This results in :
 - ◇ Systemic congestion .
 - ◇ LCOP .

Clinical picture :

Symptoms : **2 hemodynamic + 2 P**

- 1- Symptoms of systemic congestion .
- 2- Symptoms of LCOP .
- 3- **Pain** : dull aching pain due to stretch of parietal pericardium .

4- **Pressure manifestations :**

- On lung : dyspnea , improved by sitting up & leaning forward .
- On esophagus : dysphagia .
- On left recurrent laryngeal nerve : hoarseness of voice .

Signs : **2 hemodynamic + 2 P**

1- **Signs of systemic congestion :**

i - Neck vein :

- Congested neck vein .
- Kussmaul's sign : inspiratory filling of neck vein due to failure of the heart to accept the increased VR during inspiration .
- Friedreich's sign : Rapid deep Y wave . (Y wave = atrial emptying)

ii – Enlarged tender liver .

iii – Ascites before edema LL (*ascites precox*) due to :

- Kinking of hepatic vein .
- Obstruction of lymphatics passing through central tendon of diaphragm causes accumulation of lymph in peritoneum .

2- **Signs of LCOP** : cold hand , pallor , los systolic blood pressure , weak pulse .

3- **Prayer's position** .

4- **Pulsus paradoxus** : exaggerated inspiratory fall in systolic BP (>10 mmHg) .

Normally , there is slight fall in systolic BP during inspiration (< 10 mmHg)

Notice that Pulsus paradoxus is an exaggerated of the normal & not a paradox .

Explanation :

Normally : During inspiration :

- i - $\uparrow$ VR to the right side of the heart $\rightarrow \uparrow$ COP .
- ii – Expansion of the lung $\rightarrow$ taking part of COP of the right ventricle.

So, the net result is : normal or Minimal $\downarrow$ of COP .

In pericardial effusion : During inspiration :

- i - $\uparrow$ VR but the compression of the right side of the heart prevents the proper filling of ventricle $\rightarrow$ no $\uparrow$ COP.
- ii – Expansion of the lung $\rightarrow$ taking part of COP of the right ventricle.

So, the net result is massive $\downarrow$ COP.

Causes of pulsus paradoxus :

- 1- Pericardial effusion .
- 2- Constrictive pericarditis .
- 3- Restrictive cardiomyopathy .
- 4- COPD .
- 5- Acute severe asthma.

Cardiac examination :**Inspection & palpation :**

- Apex is not visible or palpable.
- Pericardial pulse in children.

Percussion :

- Dullness outside the apex.
- Dullness to the right border of the sternum.
- Dullness over the second space (disappears on sitting).
- Wide bare area.
- Dullness below the left scapula due to compression of the left lung (Ewart's sign)

Auscultation : weak distant heart sounds.

Complications :

- 1- Cardiac tamponade .
- 2- Constrictive pericarditis .

Investigations :

- **X ray** : Flask shaped heart (narrow base) with sharp border .
- **ECG** : low voltage .
- **Echo** : The most important for diagnosis & for detection of severity.
- **Catheterization** : Not needed any more after echo .
- **Aspiration (pericardiocentesis)** : may needed for diagnosis of the cause by analysis of the pericardial fluid.

DD of pericardial effusion :

- Right sided heart failure .
- Constrictive pericarditis .
- Restrictive cardiomyopathy .
- TS & TR .
- Generalized edema : liver cirrhosis , nephrotic syndrome .

Treatment :

- 1- Treatment of the cause e.g. TB
- 2- **Pericardial aspiration (pericardiocentesis)** :
 - For of cardiac tamponade .
 - For drainage of pus & injection of antibiotics or cytotoxic drugs.
 - Site of aspiration : at the angle between xiphoid process & left costal margine.
- 3- **Surgical** : Pericardiectomy or pericardio – pleural window.

Cardiac tamponade

It's a severest form of pericardial effusion.

Etiology :

- The same as pericardial effusion.
- But , the most common causes are :
 - Idiopathic.
 - CRF.
 - Cancer.

Clinical picture :

- The same as pericardial effusion **Plus**
- **Beck's triad** :
 - Decrease of systolic BP .
 - Distended Jugular vein .
 - Diminished heart sounds (quite heart) .

Investigations & treatment : The same as pericardial effusion .

3- Constrictive pericarditis

Fibrosis & marked adhesion between the 2 layer of the pericardium.

Etiology :

- The same as dry pericarditis with exception of rheumatic fever.
- The most common cause is TB & irradiation.

Hemodynamics :

The same as pericardial effusion with the following exception :

- The ventricular filling is reduced in late diastole when the elastic limit of the pericardium is reached.
- **Calcification** of pericardium is common.

Clinical picture :**Symptoms :**

Similar to pericardial effusion but :

- Without or with minimal pressure manifestations.
- AF in 30 % of cases.

Signs :

Similar to pericardial effusion but with no special decubitus (*no prayer's position*)

Cardiac signs :

- Weak or absent apical pulsation.
- **Pericardial knock** : High pitched early diastolic sound due to sudden halting of the relaxing ventricles by rigid pericardium.

Investigations :

- **X ray** : Calcification.
- **ECG** : Low voltage , Flat or inverted T wave .
- **Echo** : Pericardial thickening , calcification , exclude the effusion.
- **CT & MRI** : Highly diagnostic.

Treatment :

- Pericardiectomy.
- Treatment of the cause e.g. TB. & complications e.g. AF.

4- Adhesive pericarditis

- Adhesions between the pericardium & the surrounding mediastinal strictures & chest wall.
- It occurs as a late complication of rheumatic fever or may be due to an extension of inflammation from neighbor strictures.

9

Systemic hypertension

Definition:

Persistent elevation of arterial BP $\geq 140/90$ mmHg & above $130/80$ mmHg in the patients with diabetes or renal disease. (at least 3 times with some weeks apart or one reading in addition to 1 target organ damage or blood pressure $\geq 170/110$).

Classification of hypertension :

Stage	Systolic BP (mmHg)	Diastolic BP (mmHg)
<i>Optimal</i>	< 120	< 80
<i>Normal</i>	120 - 130	80 - 85
<i>High normal (Prehypertension)</i>	130 - 140	85 - 90
<i>Grade I</i>	140 - 160	90 - 100
<i>Grade II</i>	> 160	> 100
<i>Grade III</i>	> 180	> 110
<i>Isolated systolic hypertension</i>	> 140	< 90

Types:**1- Isolated systolic hypertension :**

- ↑ systolic BP (> 140 mmHg) without ↑ diastolic BP.
- Etiology :
 - Atherosclerosis : ↓ aortic compliance.
 - ↑ stroke volume : hyperdynamic circulation , AR , PDA.

2- Systolic & diastolic hypertension :

↑ of both systolic & diastolic BP, this is the true hypertension.

I - Primary (essential) hypertension :

- It represents approximately 95% of all cases.
- It has no known cause.
- Age of onset : usually at early middle age > 35 years.
- +ve family history.

- Predisposing factors : Genetic, obesity , Stress , Salt sensitivity, Smoking.

- Theories :

- 1- Sympathetic over activity.
- 2- Activation of the renin system.
- 3- Increased adrenal gland activity → ↑ aldosterone secretion.
- 4- Multifactorial theory :
Stress → ↑ sympathetic → renal ischemia → ↑ rennin → ↑ aldosterone → ↑ BP.
- 5- Hyperinsulinemia due to peripheral insulin resistance.
- 6- Decreased atrial natriuretic peptide (ANP)
- 7- Baroreceptors resetting.

II – **Secondary hypertension** : (curable hypertension)

- Hypertension with a known underlying cause.
- It represents approximately 5% of all cases.
- Secondary hypertension is suspected when the patient has any of the following :
 - a. Age of onset : before 25 or after 55 years.
 - b. -ve family history.
 - c. Rapidly progressive hypertension with early complications.

1- Renal : *Details (hypertension & kidney) : see below*

i – Parenchymal : (volume dependent hypertension)

GN , diabetic nephropathy , pyelonephritis , polycystic kidney ,

Mechanism :

- Ineffective in disposing Na.
- Fail to produce necessary VD substances (PG).

ii – Reno-vascular : renal artery stenosis which by turn activate the renin system.

C/P : Generalized atherosclerosis & flank bruits.

2- Endocrinal :

- Pituitary : Acromegaly (endothelial hyperplasia , Na & water retention)
- Thyroid : - **Hypothyroidism**.
- Hyperthyroidism → isolated systolic hypertension.

- Parathyroid : Hyperparathyroidism.
- DM.
- SRG :
 - ✓ Conn's syndrome : never sever HTN ,muscle weakness & hypokalemia.
 - ✓ Cushing syndrome.
 - ✓ Pheochromocytoma : paroxysmal HTN.

3- CNS :

- ↑ ICT .
- Lesions of the medulla.

4- Vascular :

- Polyarteritis nodosa .
- Polycythemia .
- Coarctation of the aorta.

5- Iatrogenic :

- **C**ontraceptive pills.
- **C**ortisone.
- **C**atecholamine.
- **C**alcium.
- **C**yclosporine (Immunosuppressant drug).

Clinical picture:

Symptoms :

- 1- Asymptomatic in most cases.
- 2- May discovered accidentally.
- 3- Headache after information.
- 4- Headache is usually occipital.
- 5- Blurring of vision , tinnitus , epistaxis , nausea & vomiting.
- 6- Complications of HTN may be the first presentation.

Signs : (Examination)**➤ Blood pressure :**

- **persistent** elevation $\geq 140/90$ mmHg & above $130/80$ mmHg in the patients with diabetes or renal disease.
 - ✓ At least 3 times with some weeks apart ... or
 - ✓ One reading in addition to 1 target organ damage ... or
 - ✓ Blood pressure $\geq 170/110$
- It should be measured in both arms (a significant difference may suggest aortic dissection).

➤ Signs suggesting 2ry hypertension : e.g.

- Auscultation of abdominal murmurs (reno-vascular hypertension).
- Auscultation of precordial or chest murmurs (aortic disease)
- Palpation of enlarged kidneys (polycystic kidney).
- Features of Cushing syndrome.

➤ Signs of complications (organ failure) : e.g.

- Ophthalmoscope examination : The presence of new retinal hemorrhages, exudates, or papilledema suggests hypertensive retinopathy.
- Neurological examinations : e.g. motor or sensory defects, signs of lateralization.
- Cardiac examination : e.g. jugular venous distention, gallop, crepitations, and peripheral edema.
- Peripheral arteries : absence, reduction, or asymmetry of pulse.

➤ Auscultation of systemic hypertension :

- Accentuated S₂
- Closed splitting S₂
- S₄
- Ejection click.
- Ejection systolic murmur : due to relative AS.
- Early diastolic murmur : due to relative AR.

Resistant hypertension:

It is defined as persistent elevation of BP in spite of use of triple antihypertensive drugs including diuretics for > 2 months.

Hypertensive urgency:

Rapid rise of BP > 220/120 mmHg & not associated with target organ damage e.g. renal failure, heart failure, stroke

Hypertensive emergency:

Rapid rise of BP > 220/120 mmHg & associated with target organ damage (TOD).

Accelerated HTN : Hypertensive crisis with grade III retinopathy.

Accelerated : Accelerated → grade 3 retinopathy



Malignant HTN : Hypertensive crisis with grade IV retinopathy plus papilledema.

Complications :**1- Cardiac :**

- **LSHF :** due to pressure overload .
- **Ischemic heart disease :** due to atherosclerosis & hypertrophy.
- **Bernheim effect :** (signs of RSHF)

Hypertrophy of LV may cause bulging of the septum into the RV → leading to slight impairment of the filling of RV → signs of RSHF.

2- Cerebral :

- Cerebral atherosclerosis.
- Cerebral stroke (ischemic or hemorrhagic stroke).
- **Hypertensive encephalopathy :**

As a result of acute rise of BP, the cerebral blood vessels are no longer able to maintain the necessary degree of constriction (failure of auto regulation) & they begin to dilate → ↑ cerebral blood flow → ↑ ICT, brain edema, coma & convulsion may occur.

NB

How to differentiate between stroke & hypertensive encephalopathy ?

- **Stroke** : Signs of lateralization (unilateral)
- **Hypertensive encephalopathy** : No signs of lateralization (bilateral)

3- Renal :

- Renal failure.
- Hematuria & proteinuria.

4- Retinal : 4 grades

- Grade I : Thickening of retinal arterioles (*silver wire appearance*).
- Grade II : Kinking of retinal veins.
- Grade III : Hemorrhage & exudates.
- Grade IV : Papilledema.

5- Vascular :

- Atherosclerosis .
- Aortic dissection.

Investigations :**1- Routine tests :**

- CBC.
- Plasma glucose.
- Serum cholesterol, uric acid, K, creatinine.

2- Investigations for complications :

- Cardiac : X ray, ECG, Echo,
- Cerebral : CT, MRI brain .
- Renal : urine analysis, renal function, renal imaging.

3- Investigations for the cause :

When secondary HTN is suspected or in a case of refractory hypertension e.g. measurement of rennin, aldosterone, catecholamines, adrenal US, brain MRI

Treatment :

The target BP is lower than $140/90$ mmHg, unless the patient has diabetes or renal disease, in which case the target would be lower than $130/80$ mmHg.

A) Lines of treatment :

I – Non pharmacological (*lifestyle modification*).

II – Pharmacological :

- Treatment of associated risk factors e.g. hyperlipidemia
- Treatment of the cause : in a case of secondary hypertension.
- Antihypertensive drugs.

B) Choice of treatment.**A) Lines of treatment :****I – Non pharmacological (lifestyle modifications):**

- Lose weight if overweight.
- Reduce salt intake.
- High K & Ca intake.
- Reduce dietary fat intake.
- Stop smoking.
- Regular exercise.
- Avoid stressful condition as possible (meditation).

Value :

- ✓ May normalize BP in prehypertension or in mild cases without any drug.
- ✓ Facilitate BP control by antihypertensive drugs.
- ✓ Control of risk factors.

II - Antihypertensive drugs :

1. Diuretics

- Types, action, side effects : Refer to heart failure.
- **Thiazide** is most commonly used in the treatment of hypertension.
- Lasix is not routinely used in a stable cases of hypertension.
- Indapamide (*natrilix*) : thiazide analogue which has dilator effect with minimal diuretic effect.
- K sparing diuretic is often used with thiazides (*Aldactazide, Moduretic*)

2. Sympathetic blockers

Centrally acting : **Clonidine** (*Catapress*)

- **Action** : stimulation of α_2 adrenergic receptors which are sympatho-inhibitors .
- **S/E** : ☒ **Rebound hypertension** with sudden withdrawal .
 - ☒ Postural hypotension.
 - ☒ Dry mouth.

Ganglion blockers : **Trimethaphan**.

Nerve ending blockers :

i. **α methyl dopa** (*Aldomet*) :

- **Action** : ↓ synthesis of catecholamines ,also has central inhibiting action .
- **S/E** : Postural hypotension , Hepatitis , Hemolysis . (**3 H**)

ii. **Reserpine** (*Brinerdin*) :

- **Action** : ↓ **Reuptake** of catecholamines (↓ stores)
- **S/E** : Depression , Nasal congestion , hypertonia.

α blockers : Prazosin (minipress)

👉 **Action** : vasodilatation .

👉 **S/E** : First dose syncope , tachycardia .

 β blockers :

👉 **Mechanism of action** :

- Is still questionable .
- $\downarrow$ contractility , $\downarrow$ HR $\rightarrow$ $\downarrow$ COP .
- $\downarrow$ renin release.

👉 **Preparation** :

- ✎ Propranolol (*indral*) : non selective β blocker.
- ✎ Atenolol (*ateno*, Tenormin), Metoprolol (*betaloc*) , Bisoprolol (*concor*) :
Selective β_1 blockers.
- ✎ Carvedilol (*cardiol*), Labetalol : Combined β & α blockers (β blockers with vasodilation).

👉 **Side effects** :

- ☠ Lung : Bronchospasm.
- ☠ Heart : Bradycardia, Heart block.
- ☠ Depression, Impotence.

👉 **CVS uses of β blockers** :

- ☞ Hypertension.
- ☞ Angina
- ☞ Heart failure.
- ☞ Arrhythmia
- ☞ F₄
- ☞ Mitral valve prolapse.

3. Vasodilators

They are *classified* into:

Arteriolar	Venous	Both
◆ Hydralazine ◆ Minoxidil ◆ Diazoxide	Nitrates	◆ ACEIs. ◆ Na nitroprusside.

Hydralazine : (Apresoline) *used in hypertensive encephalopathy by infusion.*

S/E : ☠ Reflex tachycardia, so it is almost always administered in combination with β blocker.

☠ Precipitation of angina.

☠ Lupus like syndrome.

Minoxidil : *not used*

☠ **S/E :** The same as hydralazine + hypertrichosis ($\uparrow$ growth of body hair)

Diazoxide :

○ 100- 300 mg IV rapidly in hypertensive encephalopathy.

○ **S/E:** hyperglycemia.

Na nitroprusside : (Nipride)

☠ used in hypertensive encephalopathy, 0.5 – 2 $\mu\text{g/kg/min}$ (infusion)

☠ **S/E :** Cyanide toxicity (antidote is Na thiosulfate).

4. Calcium channel blockers

Drugs , mechanism of action , side effects : see angina.

5. Angiotensin converting enzymes inhibitors (ACE inhibitors)

These drugs inhibit the angiotensin converting enzyme which converts angiotensin I into angiotensin II, These drugs also diminish the rate of bradykinin inactivation.

Decreased angiotensin II $\left\{ \begin{array}{l} \text{VD.} \\ \downarrow \text{secretion of aldosterone} \rightarrow \downarrow \text{retention of Na.} \end{array} \right.$

Decreased bradykinin inactivation $\rightarrow \uparrow$ bradykinin which is vasodilator.

Short acting : Captopril (*capoten*) : $\frac{1}{2}$ - 2 tablet t.d.s. (tab = 25 mg)

Long acting : 1 tab / day Enalapril (*Ezapril*) , Lisinopril (*Zestril*) , Ramipril (*Tritace*).

S/E :

- ☒ Dry cough .
- ☒ Hyperkalemia .
- ☒ Skin rash.
- ☒ First dose phenomenon.

6. Angiotensin II receptor blockers (ARBs)

- ☒ Losartan (*CozAAr*)
- ☒ Valsartan (*Tareg*)
- ☒ Irbesartan (*Aprovel*)

S/E : Similar to ACE inhibitors but no cough.

Generic name	Brand name	Usual dose
I. Diuretics :		
Thiazide diuretics		
Hydrochlorothiazide	Hydrex	25 mg/d
Indapamide	Natrilix	2.5 mg/d
Loop diuretics : Furosemide	Lasix	40 mg/d
K sparing diuretics		
Spironolactone	Aldactone	50 – 100 mg/d
Amiloride	Midamor	5 mg/d
Diuretic combinations :		
Thiazide + spironolactone	Aldactazide	1 x 2
Amiloride + thiazide	Moduretic	1 x 1
Furosemide + spionolactone	Lasilactone 50, 100	1 x 2
II. Sympathetic blockers :		
β blockers :		
Atenolol	Tenormin, Ateno 50, 100	50 – 100 mg/d
Bisoprolol	Concor 5, 10	5 - 10 mg/d
Metoprolol	Betaloc, Lopressor	100 – 200 mg/d
Carvedilol	Dilatrend	25 – 50 mg/d
Propranolol	Inderal	80 – 160 mg/d
Sotalol	Betacor	80 – 160 mg/d
α blockers :		
Prazosin	Minipress 1, 2	2 mg/ tds
Doxazosin	Cardura	1 mg/d
Centrally acting (α_2 agonist) :		
Methyl dopa	Aldomet 250, 500	250 – 500 mg/tds
Clonidine	Catapress 150 (μ g)	1 x 2
III. Vasodilators :		
Na nitroprusside	Niprid	Infusion
IV. Ca channel blockers :		
Nifedipene	Epilat, Adalat	30 – 60 mg/d
Diltiazem	Altiazem	120 – 240 mg/d
Verapamil	Isoptin	120 - 240 mg/d
Amlodipine	Norvasc, Alkapress, Amilo	2.5 – 5 mg/d
V. ACEIs :		
Captopril	Captopril, Capoten, Hypopress	50 mg bid
Enalapril	Ezapril, Renitec	10 – 40 mg/d
Lisinopril	Zestril 5, 10, 20	10 – 40 mg/d
Ramipril	Tritace 2.5, 5	1 x 1
VI. Angiotensin II receptors blockers :		
Valsartan	Tareg 80, 160	80 – 160 mg/d
Losartan	CozAAR	50 mg/d

B) Choice of treatment :

- Non pharmacological measures (*lifestyle modification*) should be initiated in all hypertensive patients & those with prehypertension .
- The selection of a specific antihypertensive drug should take into consideration comorbid conditions associated with hypertension as well as the patient's personal, response & financial.

1- Uncomplicated hypertension : *Stepped antihypertensive therapy*

The treatment passes in steps & if there is not an adequate response go to the next step.

Step 1 : Initiate therapy with one of the following :

ACE inhibitors or β blockers or **C**a channel blockers or **D**iuretics

Step 2 : Combination of 2 drugs of step 1 (*including diuretic*).

Step 3 : Combination of 3 drugs of step 1 (*including diuretic*).

Step 4 : Add α blocker or hydralazineto step 3

N.B : The use of lower doses of 2 or more drugs may lower BP with fewer adverse effects than the use of higher dose of a single agent .

2- Treatment of hypertension with certain concomitant diseases :**Hypertension with heart failure**

☒ Use : ACE inhibitors , Diuretics.

☒ Avoid : Ca channel blockers

Hypertension with ischemic heart disease

☒ Use: β blocker or Ca channel blocker.

☒ Avoid : Hydralazine .

Hypertension with DM

☒ Use: ACE inhibitors , Ca channel blockers.

☒ Avoid : β blockers (masking of warning signs of hypoglycemic coma).

Hypertension with renal impairment

- ✎ Use : β blockers , Ca channel blockers , Diuretics (Lasix) , α methyl dopa & ACE inhibitors but with monitoring of creatinine level .
- ✎ Avoid : ACE inhibitors in bilateral renal stenosis .

Hypertension with Asthma or COPD

- ✎ Avoid β blockers .

Hypertension with pregnancy

- ✎ Use : α methyl dopa , Ca channel blocker , Hydralazine or Labetalol .
- ✎ Avoid : ACE inhibitors , β blockers . Diuretics .

Hypertension with hyperthyroidism

- ✎ Use : β blocker .

Hypertension with peripheral vascular disease

- ✎ Use : Ca channel blockers , α blocker .
- ✎ Avoid : β blockers.

3- Hypertensive crisis :

- It is unwise to lower the BP too quickly as it may lead to organ hypoperfusion & stroke.
- Avoid initial reduction in BP more than 25 % & remember that the patients with chronic hypertension may not tolerate a normal BP so, be judicious when lowering the BP.
- The initial goal of therapy should be to achieve diastolic BP 100-110 mmHg.
- In hypertensive emergencies (with TOD) : BP control should be within 1 hour to decrease the risk of permanent damage.
- In hypertensive urgency, BP control is more slowly over days not hours.

Approach to the patient with hypertensive emergencies :

I. History :

- History of hypertension or other significant diseases.
- Medication use.
- The history should focus on the presence of TOD (target organ damage) :
 - Cardiac : chest pain may indicate MI, shortness of breath may suggest pulmonary edema, back pain may denote aortic dissection.
 - Renal : hematuria , decreased urine volume.
 - CNS : nausea, vomiting, visual changes, seizures.

II. Examination : See signs of systemic hypertension.

III. Investigations : See investigations of systemic hypertension.

Treatment :

i. Rapid acting antihypertensive drugs :

- ✗ **Na nitroprusside (Nipride)** : $0.5 - 2 \mu\text{g/kg/min}$ (infusion) .
- ✗ Nitroglycerine : $10 - 100 \mu\text{g/kg/min}$.
- ✗ Diazoxide : 100 – 300 mg rapidly IV .
- ✗ **Nicardipine** : Ca channel blocker, 5 – 15 mg/h (infusion)
- ✗ Hydralazine : 20 mg IV .
- ✗ **Labetalol** : 20 mg IV every 10 minutes until control of BP (maximum 200 mg)
- ✗ Fenoldopam : is a new dopamine receptor agonist .
- ✗ **Lasix** may be used with one of the above agents.



Never use sublingual nifedipine to reduce BP (it can cause an unpredictable drop in BP and stroke).

ii. Specific treatment :

a) Hypertensive encephalopathy :

- Anticonvulsant : Diazepam IV.
- Cerebral dehydrating measures : 25 % Mannitol infusion with lasix.
- **Labetalol**, **nicardipine**, esmolol are the preferred medications.

b) Cerebral stroke :

- The preferred medications are **labetalol** and **nicardipine**.
- Withhold antihypertensive medications unless the SBP is >220 mm Hg or the DBP is >120 mm Hg in a case of ischemic stroke.
- Details : see neurology book.

c) Acute coronary syndrome :

- For acute coronary syndrome, beta blockers, **nicardipine**, nitroglycerin are the preferred drugs.
- Note that thrombolytics are contraindicated if the BP is $>185/100$ mm Hg.

d) Acute heart failure : Treat with vasodilators in addition to diuretics.

e) Aortic dissection : Esmolol, **Labetalol**, Na nitroprusside.

f) Preeclampsia & eclampsia of pregnancy : Hydralazine, **labetalol** & **nicardipine**.

g) Malignant hypertension : **Labetalol**, **nicardipine**, nitroprusside, enalapril

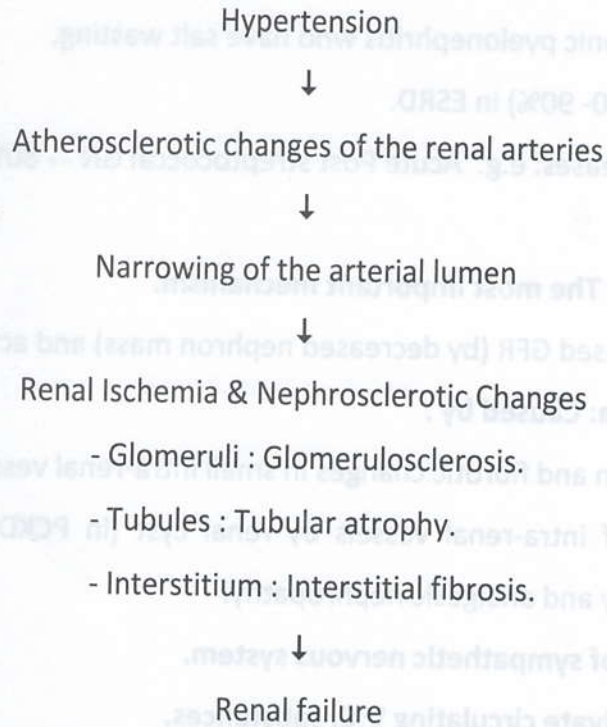
Hypokalemic hypertension :**NB**

- ➡ Conn's syndrome.
- ➡ Cushing syndrome.
- ➡ Renal artery stenosis.
- ➡ Iatrogenic : Diuretics.

Hypertension and kidney

Hypertension $\leftrightarrow$ Kidney damage

I. Effect of hypertension on the kidney :



Criteria of diagnosis of hypertensive ESRF:

- i- Duration of hypertension > 10 years before onset of R.F (Except in malignant and accelerated hypertension).
- ii- Hypertension before the onset of proteinuria.
- iii- Proteinuria < 2 gm/day.
- iv- US → No evidence of renal disease e.g. PCKD, chronic pyelonephritis, obstruction.
- v- Presence of left Ventricular hypertrophy.

II. Renal diseases as a cause of hypertension :

a). Renal parenchymal hypertension : Acute & chronic

- Ranges depending upon the stage and nature of chronic renal diseases.
- Most of renal diseases are complicated by hypertension, but exceptions are some patients with chronic pyelonephritis who have salt wasting.
- High frequency (80- 90%) in ESRD.
- In acute renal diseases: e.g. Acute Post streptococcal GN → 80%, ATN → 40%
- **Pathogenesis :** 

1- Salt retention: The most important mechanism.

Due to decreased GFR (by decreased nephron mass) and activation of RAAS.

2- Renal ischemia: Caused by :

- Inflammation and fibrotic changes in small intra-renal vessels.
- Occlusion of intra-renal vessels by renal cyst (in PCKD) or by scar in reflux nephropathy and analgesic nephropathy.

3- Stimulation of sympathetic nervous system.

4- Fails to inactivate circulating V.C. substances.

5- Fails to produce a V.D substances e.g. PGs, bradykinin.

- **Treatment :**

- Control of B.P. → slows the progression of renal parenchymal diseases.
- **Target BP :**
 - ✓ 130/80 mmHg in patients without proteinuria.
 - ✓ 125/75 mmHg in patients with proteinuria.

b). Reno-vascular hypertension : "Renal artery stenosis, RAS"

- Hypertension caused by occlusion of one or both renal arteries or their branches.
- Generally; RAS is account for $\leq 5\%$ of secondary hypertensive patients.
- The frequency increased to 75%; in elderly patients with severe hypertension and high serum creatinine.

- **Causes :**

- **The most common 2 causes are :** **95% of all causes**

1. Atherosclerosis : most common cause of RAS

- Male : Female ratio = 2: 1
- Age > 60 years.
- Usually unilateral, may be bilateral.
- Involves the proximal part of RA (near the junction of RA with aorta).
- Associated with other atherosclerotic vascular damage e.g. coronary,

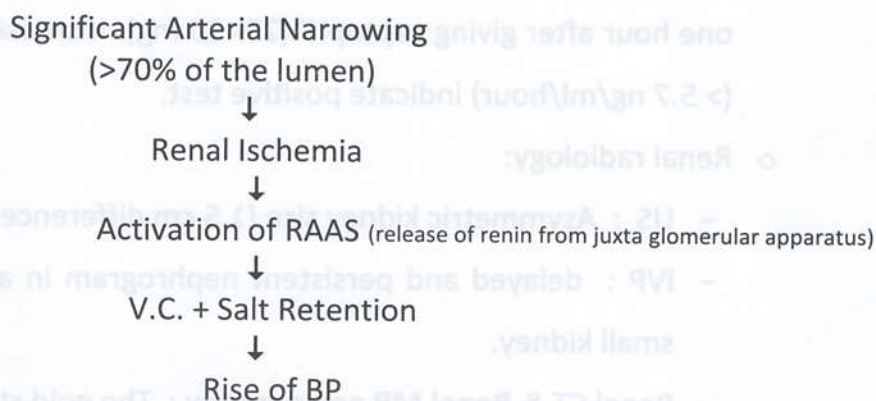
2. Fibromuscular dysplasia :

- Common in young women.
- Start unilateral then bilateral. (frequently bilateral)
- Involves the distal two-thirds of the RA. (**DYS**plasia → **Distal** 😊)
- Multifocal : beaded appearance.
- Total artery occlusion is less common than in atherosclerosis.
- Other vessel involvement e.g. cerebral vessels.

- Rare causes (5%):

- Extrinsic compression.
- Vasculitis.
- Congenital anomalies.
- Dissecting aorta.
- Embolism.
- Neurofibromatosis.

- **Pathogenesis :**



- **Diagnosis :**

- I. **Clinical diagnosis :**

- **Age :**
 - Young female (< 25 years) : Fibromuscular dysplasia.
 - Old male (> 60 years) : Atherosclerotic RAS.
 - **Hypertension.**
 - Recent onset.
 - Accelerated or malignant.
 - Recently worsen of previously controlled HTN.
 - Resistant to triple therapy including diuretics.
 - Deterioration of kidney function during treatment with ACEIs.
 - Recurrent episodes of pulmonary edema (Flash pulmonary edema) : suggest bilateral RAS.
 - Epigastric / flank bruit (especially diastolic).

- II. **Laboratory diagnosis :**

- Renal function :
 - Early in the course of the disease : normal.
 - Hypokalemia & alkalosis (due to activation of RAAS).
 - Renin activity: High, but normal values does not exclude RAS.
 - Captopril- Stimulation test : Measure the plasma renin activity before and one hour after giving captopril (25- 50 mg). Increase of plasma renin activity (> 5.7 ng/ml/hour) indicate positive test.
 - Renal radiology:
 - US : Asymmetric kidney size (1.5 cm difference).
 - IVP : delayed and persistent nephrogram in affected side + Unilateral small kidney.
 - Renal CT & **Renal MR angiography** : The gold standard for the diagnosis.

• **Treatment :**

1. Medical : β blockers, ACEIs, ARBs. Both ACEIs & ARBs are NOT allowed in a cases of bilateral renal artery stenosis, severe hyperkalemia or > 30% increase in creatinine level.
2. Renal angioplasty and stenting.
3. Renal artery bypass surgery.
4. Renal endarterectomy : Removing the fats, cholesterol and other substances (plaques) from the renal artery.
5. Nephrectomy : for small non- functioning kidney.

- **Choice of treatment:** Depends on:

- Age : - Elderly : Medical or by pass. - Young females : angioplasty.
- Severe Hypertension or renal impairment : surgery.
- Co-morbid conditions e.g. coronary, cerebrovascular : medical.
- Pathology : - Atherosclerosis : Surgery, Fibromuscular dysplasia : Angioplasty

Cardiorenal syndrome classification

Cardiorenal Syndrome (CRS) General Definition:

A pathophysiologic disorder of the heart and kidneys whereby acute or chronic dysfunction in one organ may induce acute or chronic dysfunction in the other organ

CRS Type I (Acute Cardiorenal Syndrome)

Abrupt worsening of cardiac function (e.g. acute cardiogenic shock or acutely decompensated congestive heart failure) leading to acute kidney injury

CRS Type II (Chronic Cardiorenal Syndrome)

Chronic abnormalities in cardiac function (e.g. chronic congestive heart failure) causing progressive and potentially permanent chronic kidney disease

CRS Type III (Acute Renocardiac Syndrome)

Abrupt worsening of renal function (e.g. acute kidney ischaemia or glomerulonephritis) causing acute cardiac disorder (e.g. heart failure, arrhythmia, ischemia)

CRS Type IV (Chronic Renocardiac Syndrome)

Chronic kidney disease (e.g. chronic glomerular or interstitial disease) contributing to decreased cardiac function, cardiac hypertrophy.

CRS Type V (Secondary Cardiorenal Syndrome)

Systemic condition (e.g. diabetes mellitus, sepsis) causing both cardiac and renal dysfunction

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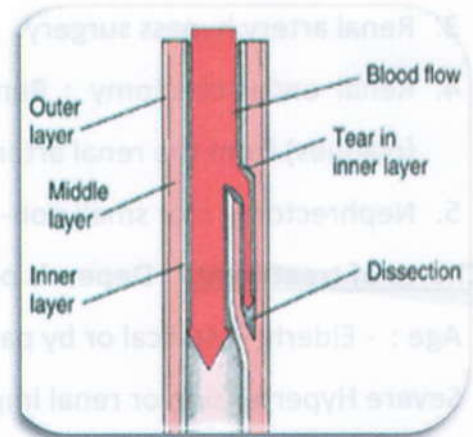
Aortic dissection

(Dissecting aortic aneurysm)

Definition:

It is a tear of the inner wall of the aorta (intima) , creating a false lumen for blood flow longitudinally along the media of the aorta.

- It is most often seen in men 50 to 70 years old.



Etiology :

- Hypertension.
- Marfan syndrome.
- Atherosclerosis.
- Trauma : e.g. during catheterization.

Classification :

- Type I - Originates in ascending aorta, propagates at least to the aortic arch.
- Type II – Originates in and is confined to the ascending aorta.
- Type III – Originates in descending aorta.

Clinical picture :

1- Chest pain : *The main symptom*

- Acute onset , Severe sharp tearing pain , radiating to the back.
- The condition must be differentiated from other causes of acute chest pain especially **MI** by the following criteria :
 - i. Patients usually have no previous attacks of similar pain.
 - ii. The pain is usually of maximal intensity from the start.
 - iii. Associated AR or unequal pulse.

The use of anticoagulants or thrombolytic therapy in a patient with aortic dissection in one word is fatal.

2- Hypertension in most cases. Hypotension may suggest aortic rupture .

3- AR in more than 50 % of cases.

4- **Obstruction of the opening of aortic branches:** depends on which arteries are blocked.

- Coronary artery $\Rightarrow$ Coronary insufficiency .
- Subclavian artery $\Rightarrow$ Unequal pulse.
- Ischemic symptoms of the brain $\Rightarrow$ Stroke , Syncope.
- Ischemic symptoms of the spinal cord $\Rightarrow$ paraplegia.
- Mesenteric arteries $\Rightarrow$ abdominal pain.
- Renal artery $\Rightarrow$ renal failure.

5- Rupture aneurysm into pericardium $\Rightarrow$ Cardiac tamponade.

6- **Sudden death** : 40 % of cases.

Investigations :

- X ray : Mediastinal widening.
- ECG : to exclude other causes of acute chest pain e.g. MI.
- Trans Esophageal Echo (TEE) , Aortography.
- **CT, MRI** : gold standard for final diagnosis.

Treatment :

1- **Control of hypertension** : β blocker (*Esmolol*) with Nitroprusside.

2- **Surgical correction** :

- Acute dissection in ascending aorta is a surgical emergency.
- Prognosis without surgery is very poor.

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Cardiomyopathy

Definition:

cardiac muscle disease in absence of known primary cardiac etiology (no hypertension , congenital , valvular , coronary or pericardial diseases)

Clinical classification :

The 3 main types of Cardiomyopathy are :

- 1- Dilated (*congestive*) cardiomyopathy (DCM).
- 2- Hypertrophic (obstructive) cardiomyopathy (HCM , HOCM , IHSS).
- 3- Restrictive cardiomyopathy.



Dilated cardiomyopathy



Hypertrophic cardiomyopathy



Restrictive cardiomyopathy

Dilated cardiomyopathy

- It is characterized by ventricular chamber dilatation & systolic dysfunction leading to congestive heart failure *hence the name* , dilated (or congestive) cardiomyopathy.
- It is the most common type ,may affect any age & sex but most often in middle age men.

Etiology :

- Idiopathic.
- Infection : viral (e.g. coxsackie B & HIV) , bacterial.
- Immunological : SLE .
- Iatrogenic : Adriamycin , Alcohol , Cocaine.
- Infiltration : Amyloidosis , Sarcoidosis , hemochromatosis.

- Endocrinal : DM , Acromegaly .
- Neurological : Freidreich's ataxia , Duchenne myopathy , myotonia atrophica . **MCQ**

Clinical picture :

- Congestive heart failure. (LSHF , RSHF)
- Arrhythmia.
- Thromboembolism.

Investigations :

- X ray , ECG , Echo : show dilated heart.
- Biopsy : for amyloidosis or sarcoidosis.

Treatment :

- Treatment of HF : ACEI , β blocker , Diuretic & heart transplant in refractory cases.
- Treatment of arrhythmia .
- Treatment of thromboembolism : anticoagulant.

Hypertrophic Cardiomyopathy

- Asymmetrical ventricular hypertrophy especially in the interventricular septum & LV.
- This thickening of myocardium results in diastolic dysfunction due to myocardial stiffness
- Also , it may result in an obstruction in the LV outflow tract $\Rightarrow$ subvalvular AS .
- Hypertrophic cardiomyopathy is considered to be the most common cause of sudden death in young athletes. **MCQ**

Etiology :

Autosomal dominant disease

- Idiopathic Hypertrophic Sub-aortic Stenosis (IHSS)
- Associated with Freidreich's ataxia.

Clinical picture :

- Diastolic dysfunction : Pulmonary congestion & LCOP.
- Manifestations of AS : Angina , Syncope.
- Sudden death : usually associated with sports most probably due to ventricular arrhythmias.

General signs :

- Giant (a) wave if there is right ventricular outflow tract obstruction.
- Jerky (bifid) carotid pulse.

Cardiac examination :

- Double apical impulse : due to palpable atrial systole (S_4).
- Paradoxical splitting of S_2
- No click as obstruction is subvalvular and not valvular.
- Left parasternal late ejection systolic murmur.
- Associated **MR** is common : apical pansystolic murmur.
- The murmur of HCM , contrary to valvular AS , decreases by leg raising or squatting because of $\uparrow$ venous return which fills the ventricle and so decrease obstruction.

Investigations :

- X ray : No cardiomegaly , pulmonary congestion.
- ECG : Left ventricular hypertrophy , deep wide Q wave , arrhythmias.
- **Echo** : Ventricular hypertrophy , mitral systolic anterior motion (SAM)

Treatment :

- Amiodarone for arrhythmias.
- β blockers : $\downarrow$ contractility & outflow obstruction.
- Ca Channel blockers : $\downarrow$ contractility & outflow obstruction.
- Avoid digitalis & other inotropics $\Rightarrow \uparrow$ outflow obstruction .
- Surgical resection of hypertrophied septum.

Restrictive cardiomyopathy

- It is characterized by restrictive diastolic ventricular filling with normal systolic function.
- It is the least common form of cardiomyopathy.

Etiology :

- Idiopathic endomyocardial fibrosis.
- Infiltration : Amyloidosis , Sarcoidosis , Hemochromatosis. 🎵🎵

Clinical picture :

- Similar to constrictive pericarditis (IF RV is involved)
 - Systemic congestion.
 - LCOP.
 - AF.
- Pulmonary congestion if LV is involved.

Cardiac signs :

- Signs of RVE : Late & mild.
- Murmur of mitral & tricuspid regurge : due to involvement of the papillary muscles by fibrosis.
- No pericardial knock.

Investigations :

- X ray : No or mild RVE , no calcification (*to differentiate it from constrictive pericarditis*)
- ECG : Low voltage.
- Echo : Diastolic dysfunction.
- Endomyocardial biopsy : Diagnostic.

Treatment :

- Treatment of the cause e.g. Hemochromatosis.
- Symptomatic treatment : HF , Arrhythmias , Thromboembolism.
- Cardiac transplant in refractory cases.

Medicine cannot, except over a short period, increase the population of the world.

Bertrand Russell

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Pulmonary circulation

Pulmonary hypertension

Definition:

Elevation of the pulmonary arterial pressure above $30/15$ mmHg. (MPA pressure > 20)

Etiology :**Primary pulmonary hypertension (PPH) :**

- Very rare (*represents less than 1 % of all cases of pulmonary hypertension*).
- Etiology is **unknown** , occurs more commonly in middle aged female with repeated pregnancies.
- May be due to repeated thromboembolism , pulmonary vasculitis.

Secondary pulmonary hypertension :**1- Hyperdynamic pulmonary hypertension :**

Due to increased pulmonary arterial blood flow :

- ✎ Hyperdynamic circulation.
- ✎ Congenital heart diseases : ASD , VSD , PDA .

2- Passive pulmonary hypertension :

Due to pulmonary congestion :

- ✎ Mitral valve disease .
- ✎ LSHF.

3- Vasoconstrictive (reactive) Pulmonary hypertension :

Due to reflex VC of pulmonary arteriole in response to :

- ✎ Long standing pulmonary congestion.
- ✎ Long standing hyperdynamic pulmonary hypertension .
- ✎ Hypoxia : COPD.

4- Obstructive pulmonary hypertension :

Long standing vasoconstrictive pulmonary hypertension → Irreversible VC of pulmonary arterioles.

5- Acute obstructive pulmonary hypertension :

Massive pulmonary embolism.

Clinical picture :

- 1- C/P of the cause .
- 2- Right ventricular enlargement .
- 3- RSHF .

4- Signs of pulmonary hypertension :**General :**

Giant "a" wave . (no giant "a" wave in ASD ??)

Cardiac :

- Inspection : Pulsation in the 2nd left intercostals space .
- Palpation : Diastolic shock (palpable accentuated pulmonary component of S_2)
- Percussion : Dullness in the 2nd left intercostals space .
- Auscultation :
 - Accentuated S_2 .
 - S_4 .
 - Ejection click .
 - Ejection systolic murmur : due to relative PS .
 - Early diastolic murmur : due to relative PR .

Investigations :**X ray :**

- Dilated pulmonary artery .
- RVE .

ECG :

- Peaked P wave (P pulmonale) .

Echo :

- Detection of the cause .
- Estimation of the pulmonary artery pressure .
- RVE.

Catheterization :

- Detection of the cause .
- Estimation of the pulmonary artery pressure .
- RVE .

Treatment :

- Treatment of the cause.
- Treatment of RSHF.
- **Drugs :** (*limited benefit*)
 - ACE inhibitors , Anticoagulants.
 - PGI_2 . (*potent systemic & pulmonary vasodilator*)
 - Ca channel blockers.
 - Diuretics.
- **Heart lung transplantation** : is the chief therapeutic option.

Pulmonary embolism

Etiology : (source of emboli)

1- **Deep venous thrombosis (DVT) :** Commonest cause (90 %)

➤ **Predisposing factors of DVT :** (Virchow triad)

- i. Slow circulation. ii. Injury of endothelium. iii. Hypercoagulability.

➤ **Risk factors for DVT :** 6 " O "

- ✗ **Operative :** especially pelvic & bone surgery .
- ✗ **Oncology :** Cancer stomach , leukemia & lymphoma .
- ✗ **Old age .**
- ✗ **Oral contraceptive pills** ✗ **Obesity.**
- ✗ **Others :** 2 "C" - Congestive heart failure
- Coagulation disorders : protein C & S deficiency .

➤ **Sites of DVT :** Deep veins of the leg particularly the iliac, femoral & popliteal veins .

2- **Detached thrombi from right side of the heart :**

Infective endocarditis , Atrial fibrillation , Myocardial infarction.

3- **Paradoxical embolism :** from the left side of the heart if there is left to right shunt:VSD

4- **Rare :** Fat embolism , Air embolism , Amniotic fluid embolism .

Clinical picture :

- Unfortunately, there are no clinical or laboratory findings that will confirm or exclude the diagnosis of pulmonary embolism .
- The clinical picture of pulmonary embolism is not specific , so successful management of pulmonary embolism requires a combination of clinical suspicion & appropriate use of diagnostic tools .
- The clinical presentations are ranging from asymptomatic to sudden death depending on the size of the embolus .

1- Small sized emboli :

- Recurrent small pulmonary emboli results in occlusion of large number of pulmonary arterioles.
- Usually asymptomatic , but non specific symptoms of tachypnea , dyspnea, tachycardia or pleuritic chest pain may occur .
- May result in the development of pulmonary hypertension & subacute cor-pulmonale over years .

2- Medium sized emboli : (pulmonary infarction)

If occlusion involves less than 60% of the vascular bed .

C/P : 

- ✎ Pleuritic chest pain.
- ✎ Cough & hemoptysis.
- ✎ Dyspnea .

3- Acute massive pulmonary embolism : occlusion > 60% of the vascular bed

- i. **Acute dyspnea .**
- ii. **Acute chest pain :** (*similar to angina pain*) due to :
 - Distension of the pulmonary artery .
 - Reflex coronary spasm .
- iii. **Sudden appearance of pulmonary hypertension .**
- iv. **Acute right sided heart failure .** (*see HF*)
- v. **Shock :** It is an obstructive shock in which there is marked ↓ of blood flow to the lung → ↓ blood flow to left atrium → ↓ COP → shock .
- vi. **Sudden death :** may be the first manifestation if occlusion involves more than 80% of the vascular bed .
- vii. Finding suggestive for DVT : Tenderness , swelling , redness of the LL .
unfortunately, DVT of the leg is often clinically silent .

Differential diagnosis :**Causes of acute dyspnea with acute chest pain :**

- 1- Pulmonary embolism .
- 2- Myocardial infarction .
- 3- Pericardial effusion .
- 4- Acute pulmonary edema (acute heart failure) .
- 5- Pneumonia .
- 6- Tension pneumothorax .
- 7- Bronchial asthma .

Clinical & Lab findings in patients with suspected pulmonary embolism : Not to memorize

Findings	+ve predictive value	-ve predictive value
Dyspnea	37 %	75 %
Tachycardia	47 %	86 %
Pleuritic chest pain	39 %	71 %
Hemoptysis	32 %	67 %
Hypoxemia	34 %	70 %
Elevated plasma D - dimer	27 %	92 %

+ve predictive value : It expresses the likelihood that a PE is present when the finding is present.

-ve predictive value : It expresses the likelihood that a PE is not present when the finding is also not present .

The ICU Book , 3rd edition 2007 Paul Marino

Investigations :**1- Chest x ray :**

- Normal in most cases .
- Elevated cupola of the diaphragm.
- Pleural effusion .
- Pulmonary infarction : wedge shaped opacity .
- Dilated pulmonary artery with decreased pulmonary vasculature (*Westermarck sign*)
- **Hypoxia with a normal x ray should raise the possibility of PE .**

2- ECG :

- May be normal .
- Sinus tachycardia & atrial fibrillation .
- $S_1 Q_3 T_3$ pattern (*S wave in lead I , Q wave in lead III & an inverted T in lead III*)
- The most important value is to exclude MI as a cause of dyspnea & chest pain.

3- Echo : Enlargement of RA , RV & pulmonary artery .

4- Pulmonary angiography : is the gold standard for making a diagnosis .

5- Spiral CT angiography : less invasive than standard angiography .

6- Ventilation – perfusion scan : Areas of normal ventilation & hypoperfusion .

7- Laboratory :

- Arterial blood gases : hypoxia , hypocapnia .
- Elevated serum LDH .
- D – Dimer : (*an end product of fibrin digestion*)

It is a good –ve test : *if D-dimer is not elevated , PE is unlikely .*

8- Investigation for diagnosis of DVT : Duplex ultrasound .

Treatment of pulmonary embolism & DVT :

I- Prophylaxis :

1. Early post operative ambulation .
2. Elastic stocking .
3. Elevation of the leg .
4. Anticoagulants in high risk patients : *for about one week*
 - ✗ Low dose heparin 5000 units SC / 8-12 hr .
 - ✗ Low Molecular Weight heparin (enoxaparine : *Clexane*) 20-40mg/d.

II- Curative :

- 1- Hospitalization in CCU .

- **Heparin :**

- tion : activation of antithrombin III .

- **LMW heparin :** 1mg/kg SC/12 h. No need for routine anticoagulant monitoring

➤ **Oral anticoagulant :** warfarin (Marevan)

- n K $\rightarrow$ ↓ formation of

3- Thrombolytic therapy : Streptokinase , tPA

- Indicated in life threatening massive pulmonary embolism when the patient

4- Pulmonary embolectomy :

- Is used for those with massive PE when thrombolysis is contraindicated.

5- Symptomatic treatment :

- Pain : Analgesics e.g. pethidine (*avoid morphine*)

6- Treatment of complications : - Shock

- RSHF.

7- Inferior vena caval filters : e.g. *Greenfield filter*

- Meshlike filter can be placed in the inferior vena cava to trap emboli & prevent them from traveling to the lungs .
- Should be considered when anticoagulants are contraindicated .

COR PULMONALE

Definition :

Cor pulmonale is right ventricular enlargement with or without failure resulting from pulmonary disease after exclusion of LSHF & congenital heart diseases .

Pulmonary disease → Pulmonary HTN → RV enlargement → RSHF.

Etiology :

1- **Acute cor pulmonale :**

- Acute massive pulmonary embolism .
- Acute massive lung collapse .
- Tension pneumothorax .

2- **Subacute cor pulmonale :**

- Recurrent small pulmonary embolization.
- Lymphangitis carcinomatosa of the lung .

3- **Chronic cor pulmonale :**

- Parenchymal lung diseases : **COPD** , **IPF** .
- Pulmonary vascular diseases : **Bilharziasis** , primary pulmonary hypertension .
- Thoracic wall diseases : kyphoscoliosis , Pickwickian syndrome .

Clinical picture , investigations , treatment : 4

- Of the cause : Bilharziasis :** see *GIT book p 50* , **COPD :** see chest .
- Of pulmonary hypertension .**
- Of RV enlargement :** may be masked by emphysema in a case of **COPD** .
- Of RSHF .**

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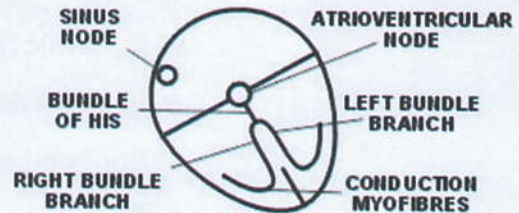
Arrhythmia

Definition:

Arrhythmia is an abnormality of the cardiac rhythm or rate.

The conducting system of the heart :

- ✓ Under normal condition ,the pacemaker of the heart is **Sinoatrial node(SAN)**
- ✓ The cardiac impulses arises from **SAN** in a rate (60 – 90 beats/min)
- ✓ The impulse spreads through the walls of the atria causing them to contract.
- ✓ Next ,the impulse reaches the **AV node** ,in which there is a delay of conduction to allow the atria to contract before the ventricles.
- ✓ Then the impulse reaches **bundle of His** in the interventricular septum , then along the **2 bundle branches** (left & right) & finally **Purkinje fibers** to terminate in the ventricular myocardium causing ventricular contraction.



- ✗ Sympathetic stimulation → ↑ the activity of SAN & ↑ the conduction of AVN .
- ✗ Parasympathetic stimulation → ↓ the activity of SAN & ↓ the conduction of AVN .
- ✗ The ventricles are supplied by sympathetic only (*no parasympathetic supply*) .
- ✗ **SAN** is considered the **pacemaker** of the heart because its normal rate (60-90b/m) is faster than other cardiac muscle fibers.
- ✗ SAN is characterized by its own automaticity (ability to generate impulses) so nerve supply of the heart aims at regulation of heart rate & not initiation of rhythm.
- ✗ Normally, the AVN allows passage of impulses from atria to ventricles but not the reverse (*no retrograde conduction*)

Clinical classification of arrhythmias:

➤ Regular tachycardia :

- Sinus tachycardia.
- Paroxysmal supra-ventricular tachycardia .
- Atrial flutter .
- Ventricular tachycardia .

➤ Regular bradycardia :

- Sinus bradycardia .
- Nodal (junctional) rhythm .
- Partial heart block (1st & type II 2nd degree heart block).
- Complete heart block (3rd degree heart block).

➤ Irregular rhythm :

- Premature beats (Extrasystoles).
- Atrial fibrillation .
- Type I 2nd degree heart block .

Arrhythmia Scheme

I- Etiology of any arrhythmia : 7

Tachyarrhythmia	Bradyarrhythmia
1- Myocarditis. 2- Ischemic heart disease (Myocardial infarction). 3- Rheumatic heart disease . 4- Congenital heart disease . 5- Digitalis.	
6- Sympathomimetics . 7- Thyrotoxicosis .	6- Sympatholytics 7- Hypothyroidism .

Exceptions :

- Sinus (tachy or brady) arrhythmias : Physiological & pathological causes .
- Atrial flutter or Atrial fibrillation : begin the etiology by : MS & thyrotoxicosis.

II- Clinical picture :

➤ Symptoms of tachyarrhythmias :

- 1- Asymptomatic .
- 2- palpitation :
 - Onset :
 - Offset :
 - Duration of the disease : short in serious arrhythmias e.g. VT, CHB.
- 3- Manifestations of LCOP .
- 4- Precipitation of HF & angina.
- 5- Features of the cause e.g. : MI , Rheumatic heart disease, digitalis toxicity

➤ Symptoms of bradyarrhythmias :

The same but no precipitation of angina .

Exceptions :

- Atrial fibrillation (AF) : add thromboembolism.
- Ventricular tachycardia (VT) : add Sudden death .
- Complete heart block : add Syncope , Sudden death .

Signs :**1- Radial pulse :** (test for ventricle) 4

- a) **Rate** : ↑ (Uncountable pulse) in tachy , ↓ in bradyarrhythmias .
- b) **Rhythm** : all are **regular** except AF & extrasystoles.
- c) **Response to carotid sinus massage (in tachy)** : ↓ HR in any tachyarrhythmia except arrhythmias that originate in the ventricle .

simply: any arrhythmia contain this word , **ventricular** , in its name → no effect 😊
(no parasympathetic supply)

NB : In bradyarrhythmias : Response to atropine instead .

- d) **Respiratory sinus arrhythmia**: **-ve** in all arrhythmias except in both sinus tachy & bradyarrhythmias .

Simply, it is **-ve** in any arrhythmia except when arrhythmia contains this word , **sinus** ,
→ it is **+ve** . 😊

Respiratory sinus arrhythmia : (HR is increased during inspiration)

- Inspiration → ↑ VR → ↑ of SAN → ↑ HR .
- This is a physiological process indicating that the pacemaker is the SAN .

2- Neck vein : (test for atrium)

- Rapid A wave in atrial tachyarrhythmias.
- Loss of A wave in atrial fibrillation.
- **Cannon A** wave in any arrhythmia containing this word : **nodal**, either :
paroxysmal **nodal** tachycardia or **nodal** rhythm.

- Occasional cannon A wave in : ventricular tachycardia & complete heart block
(Atrio-Ventricular dissociation).

Cannon A wave : It means severe increase of the right atrial pressure .

It is due to ventricular contraction during atrial contraction .

3- Auscultation : (first heart sound)

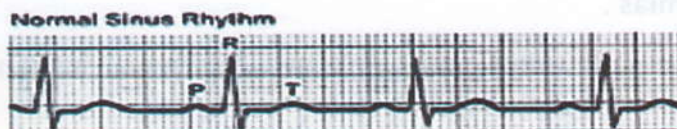
- Accentuated in any tachycardia.
- weak in any bradycardia.

Exceptions :

- Atrial fibrillation .
- Ventricular tachycardia. } Variable S₁
- Complete heart block .
- Nodal rhythm ➔ accentuated S₁ inspite of bradycardia .

III- Investigations :

1- ECG :



P wave : *represents atrial contraction .*

- Normal in sinus arrhythmias .
- Abnormal in any other atrial arrhythmias .
- Flutter wave in atrial flutter.
- Fibrillation waves or even absent P wave in atrial fibrillation.

PR interval : *represents the passage of impulse from atria to ventricles .*

- Short in tachycardia.
- Prolonged in bradycardia.
- AV dissociation in : VT , CHB .

QRS complex : *represents the ventricular contraction .*

- Regular except in AF & extrasystole.
- deformed (bizarre) : in VT & CHB .

2- Investigations for the cause :

- Echo : Congenital or valvular heart diseases.
- Thyroid function tests .

IV- Treatment :

See later

NB : This scheme is more than enough for undergraduates

Imagination is more important than knowledge.

Albert Einstein

Sinus tachycardia

Definition :

It is a condition in which the SAN discharges impulses faster than normal (>100 / min)



Notice that SAN is still the pacemaker of the heart

Etiology :

- **Physiological** : Exercise , Emotions , Excessive coffee .
- **Pathological** : Hypotension,Hyperdynamic circulation, Hyperthermia , Heart failure
- **Pharmacological** : Adrenaline , Atropine .

Clinical Picture :

Symptoms :

- The same as scheme .
- Onset & offset : gradual.
- Duration of the disease is usually long as the condition is mostly physiological.

Signs :

1- Radial pulse :

- **Rate** : > 100 /min but usually less than 160 / min.
- **Rhythm** : regular.
- **Response to carotid sinus massage** : gradual $\downarrow$ HR
- **Respiratory sinus arrhythmia** : +ve.

2- Neck vein : Normal rapid waves .

3- Auscultation : Accentuated S_1 .

ECG :

- **Rhythm** : regular.
- **Rate** : 100 – 160 / min.
- **P waves** : are normal & each P wave is followed by normal QRS .

Treatment :*usually no need*

- Treatment of the cause.
- β blockers & sedatives may be needed .

Paroxysmal supraventricular tachycardia**Definition :**

- It is a paroxysmal condition in which there is an abnormal focus in the atrium – *other than SAN* - which discharges regular impulses more than SAN (150-250/min).
- This abnormal focus may initiated in any area of the **atria** (*paroxysmal atrial tachycardia*) or even in **AVN** (*paroxysmal nodal tachycardia*).



Notice that the heart neglects the SAN & follows the focus

Etiology :

- **Physiological** : excessive coffee , smoking.
- **Pathological** : the same as scheme.

Clinical picture : (in between the attacks the heart is normal)

Symptoms :

- **The same as scheme.**
- Sudden onset & offset.

- Duration of the disease: usually long history as the condition is mostly physiological.
- Duration of the attack : Variable , usually few minutes but may lasts for hours.

NB : PSVT that lasts for more than 50 % of the day is considered a permanent PSVT.

Signs : *during the attack*

1- Radial pulse :

- **Rate :** 150 – 250 beats/min. (*uncountable*).
- **Rhythm :** regular .
- **Response to carotid massage :** sudden ↓ HR .
- **Respiratory sinus arrhythmia :** -ve . (SAN is not the pacemaker)

2- Neck vein :

- **Atrial tachycardia :** Normal rapid waves .
- **Nodal tachycardia :** Cannon A waves .

3- Auscultation : Accentuated S₁ .

ECG :

- **P wave :** - In **atrial** tachycardia : deformed.
- In **nodal** tachycardia : absent or inverted.
- **QRS :** rapid , regular with normal shape.

Treatment :

During the attack

1- Vagal stimulation : Carotid sinus massage or pressure on eye ball.

2- Drugs : A B C D

Adenosine , **β** blockers , Ca channel blockers (verapamil) , Digitalis. (IV)

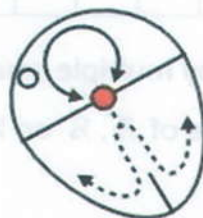
3- If there is no response or if the patient is hemodynamically unstable :

DC cardioversion.

Atrial Flutter

Definition :

It is a condition in which there is an abnormal focus in the atrium that discharges rapid regular impulses (**250 – 350 /min**) , but due to **physiological block** of AVN , not all atrial impulses are conducted to the ventricles – only $\frac{1}{2}$, $\frac{1}{3}$, $\frac{1}{4}$, ...of the atrial impulses will pass to the ventricles .



Notice that not all atrial impulses are conducted to the ventricles

Etiology : *doesn't occur in normal heart*

The same as scheme but begin with : **Mitral stenosis & thyrotoxicosis** . 🎵🎵

Clinical picture :

Symptoms :

- The same as scheme.
- Sudden onset & offset .
- Duration of the disease : Short , it is a transient arrhythmia between normal sinus rhythm & atrial fibrillation .

Signs :

1- Radial pulse:

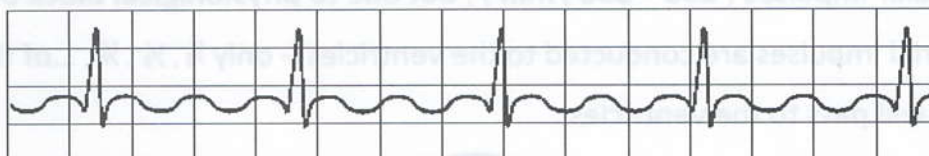
- **Rate** : Variable according the degree of AV conduction , 150 , 100, 75 beats/m.
- **Rhythm** : regular .
- **Response to carotid massage** : ↓ HR in mathematical pattern due to ↑ AV block from 2:1 to 3:1 to 4:1 So, HR ↓ from 150 to 100 to 75 beats/min .
- **Respiratory sinus arrhythmia** : -ve (SAN is not the pacemaker)

2- Neck vein : number of A waves is double, triple or quadruple the pulse rate according to the degree of AVN conduction .

3- Auscultation : Accentuated S_1 .

ECG :

(Saw tooth appearance)



- **P waves :** abnormal ,replaced by multiple small flutter (f) waves before each QRS
- **QRS :** normal , regular , at a rate of $\frac{1}{2}$, $\frac{1}{3}$ or $\frac{1}{4}$ the atrial rate according to AVN conduction.

Treatment :

1- Drugs : to control the ventricular rate ($\downarrow$ AVN conduction)

β blockers , Ca channel blocker (*verapamil*) or digitalis .

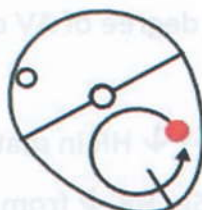
2- DC cardioversion : if the patient is hemodynamically unstable.

Ventricular tachycardia

Definition :

It is a paroxysmal condition in which there is abnormal focus in the ventricle that discharge impulses more than SAN (150 – 250 / min).

- Since the focus is in the ventricle & there is no retrograde conduction in the AVN, So ventricles will follow the ectopic focus & atria will follow the SAN (*AV dissociation*)



Notice that there is no retrograde conduction in the AVN

Etiology : occur in patient with established heart disease

- The most common cause is ischemic heart diseases (**myocardial infarction**).
- Other causes : the same as scheme .

Clinical picture :

Symptoms :

- **The same as scheme.**
- Sudden onset & offset .
- Duration of the disease : short history because it is a serious condition.
- Duration of the attack :
 - **Sustained VT** : more than 30 seconds (hemodynamically unstable)
 - **Non sustained VT** : less than 30 seconds .
- **Sudden death** : if converted to ventricular fibrillation .

Signs :

1- Radial pulse :

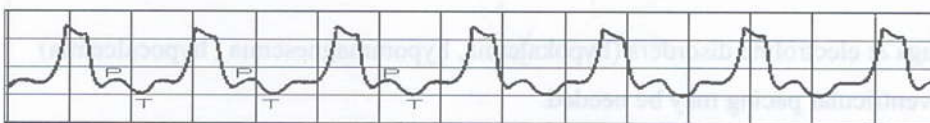
- **Rate** : 150 – 250 / min (*uncountable*).
- **Rhythm** : regular .
- **Response to carotid massage** : no effect (*no parasympathetic supply to ventricles*)
- **Respiratory sinus arrhythmia** : -ve .

2- Neck vein :

- Normal "A" wave .
- Occasional cannon A wave (*because occasionally the atria & ventricles may contract together*).

3- Auscultation : Variable S_1 , occasionally cannon sounds.

ECG :



- **QRS** : rapid, regular & **wide abnormal** (bizarre) shaped.
- **P waves** : Normal rate & shape.
 - May comes before or after the QRS and also may be hidden by the QRS.
- **No fixed relation between P waves & QRS complexes** (*atrio ventricular dissociation*)

NB: Any wide QRS complex tachycardia in any patient with primary heart disease is considered & treated as VT until proved otherwise.

Treatment :

During the attack :

- **If the patient is hemodynamically unstable :**
 - Immediate cardioversion (start at 100 J & repeat if needed & add 100 J to each successive shock.)
- **If the patient is hemodynamically stable :**
 - ✎ Amiodarone (IV) : 150 mg IV over 10min & follow with 1mg/min infusion for 6 hours.
 - ✎ Lidocaine (IV).

- Recently, amiodarone has replaced lidocaine as the antiarrhythmic drug of choice in terminating VT.
- Adenosine is not effective in VT. MCQ

In between the attacks : **albi**

- Amiodarone .
- Lidocaine.
- β blockers .
- Implantable Cardioverter defibrillator (ICD) : in resistant cases.

Torsades de points : (*French for twisting of the points*)

- It is a multifocal VT characterized by QRS complexes that change in amplitude & appear to be twisting around the isoelectric line of the ECG & associated with prolonged QT interval.
- AE :
 - Antiarrhythmic drugs & electrolyte disorders (hypokalemia, hypomagnesemia , hypocalcemia)
- Treatment : Mg & ventricular pacing may be needed.

DD of regular tachycardia

Etiology	Physiological:3E Pathological :4H Pharmacological:2A	Excessive coffee & smoking Pathological:scheme	MS Thyrotoxicosis	MI
Complaint (palpitation)	Gradual onset Gradual offset Long history	Acute onset Acute offset Long history	Acute onset Acute offset Short history (transient)	Acute onset Acute offset Short history (serious)
Radial pulse : - Rate - Rhythm ☺ - Response to carotid massage - Respiratory sinus arrhythmia	100 – 160 /m Regular +ve (gradual ↓) +ve	150 – 250 /m Regular +ve (sudden ↓) -ve	Variable(150,100,...) Regular +ve (mathematical) -ve	150 – 250 /m Regular - ve -ve
Neck vein	Rapid & normal	Atrial :rapid ,normal Nodal : cannon	Multiple a wave : 2,3,4 time the radial rate	Normal with occasional cannon
S₁	↑	↑	↑	variable
ECG	Rapid normal	Atrial : P waves are deformed QRS : normal shape Nodal : absent P wave	- P wave : flutter waves - QRS : ½, ⅓, ¼ the P waves.	Wide bizarre QRS AV dissociation.
Treatment	• ttt of the cause • β blocker	• Vagal stimulation • Drugs : A,B,C,D. • Cardioversion	• Drugs: B, C, D • Cardioversion	• Cardioversion • Amiodarone • Lidocaine.

Sinus bradycardia

Definition :

It is a condition in which the SAN discharges impulses by a rate less than 60 / min

Etiology :

- **Physiological** : During sleep , Athletes .
- **Pathological** : Obstructive jaundice , Hypothyroidism.
- **Pharmacological** : β blockers , Ca channel blockers , Digitalis .

Clinical picture:

Symptoms : usually asymptomatic

- **The same as scheme** (notice that there is no precipitation of angina)
- Onset & offset : gradual .
- Duration of the disease is usually long as the condition is mostly physiological.

Signs :

1- Radial pulse :

- **Rate** : < 60 /min.
- **Rhythm** : regular.
- **Response to exercise or atropine** : gradual $\uparrow$ HR
- **Respiratory sinus arrhythmia** : +ve .

2- Neck vein : Slow – normal shape .

3- Auscultation : Weak S_1 .

ECG :

- **Rhythm** : regular.
- **Rate** : < 60/min.
- **P waves** : are normal & each P wave is followed by normal QRS .

Treatment : *usually no need*

- Treatment of the cause.
- Atropine may be needed.
- Artificial pacemaker may be needed in severe chronic cases or when sinus bradycardia is a part of *Sick Sinus Syndrome*.

Nodal (Junctional) rhythm

Definition :

- It is a condition in which the heart is controlled by the AVN .
- Here , the impulses reach the atria & ventricles in the same time .

Etiology : The same as scheme (*the most common causes are digitalis & MI*)

Clinical picture :

Symptoms :

- **The same as scheme.**
- Sudden onset & offset .
- Duration of the disease : usually short history except if congenital .

Signs :

1- Radial pulse :

- **Rate :** slow (40 – 50 /min) .
- **Rhythm :** regular.
- **Response to exercise or atropine :** gradual ↑ HR .
- **Respiratory sinus arrhythmia :** -ve . (*SAN is not the pacemaker*)

2- Neck vein : Cannon A waves .

3- Auscultation : accentuated S₁ (cannon sounds)

This is an exception in bradyarrhythmia.

ECG :

- P wave is inverted, may be before, under or after QRS complex

- HR is slow

- **P waves** : Inverted & come approximately at the same time with QRS so may be *absent*
- **QRS** : Slow , regular with normal shape .

Treatment :

- Treatment of the cause.
- Atropine.
- Artificial pacemaker may be needed in severe cases.

HEART BLOCK

Types :

- **Sino atrial block** : failure of impulse to conduct between the SAN & the atria.
- **AV block** : failure of impulse to conduct between the atria & the ventricles.
- **Bundle branch block (BBB)** : either in left or right bundles .

Atrio ventricular (AV) block

First degree heart block : (Just delayed conduction)

- **PR interval is longer than 0.2 second.**
- All impulses from SAN are conducted to the ventricles.
- Etiology : physiologically during sleep or pathologically as in myocarditis.
- Usually asymptomatic.

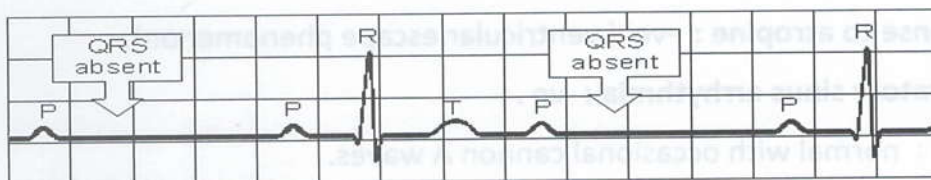
Second degree heart block :

In this condition some impulses from the atria don't reach the ventricles, this causes "**dropped beats**". There are two types :

Type I 2nd degree (Mobitz I , Wenckebach block) :

- Progressive prolongation of PR interval leading finally to the dropout of a QRS complex & then the cycle is repeated. (notice that there is irregular pulse).
- This condition is not too serious and may occur physiologically during sleep in athletes.

Type II 2nd degree (Mobitz II) :



Intermittently skipped ventricular beat

- The AVN transmits one impulse for each 2 ,3, 4 or more atrial impulses .
- This block may be fixed (e.g. 2:1 all the time) or variable (irregular).

Complete heart block (3rd degree) :

- In this condition all impulses from the atria don't reach the ventricles so, the ventricles will be controlled by **idioventricular rhythm**.



Notice that the atria are controlled by SAN & the ventricles are controlled by idioventricular rhythm.
(Atrio ventricular dissociation)

- Idioventricular rhythm may originate anywhere from AVN to the bundle branches or purkinje fibers. (*The closer the origin to AVN , the faster the rate*)

Etiology : The same as scheme plus idiopathic fibrosis of AVN.

Clinical picture :

Symptoms :

- The same as scheme. Plus **2S**
- Syncope "Adams-Stokes attacks"
- Sudden death.

Signs :

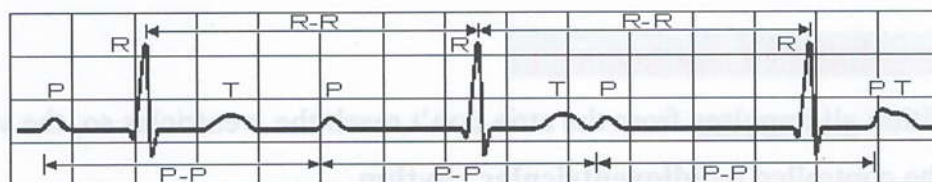
1- Radial pulse :

- Rate : 30-40 /min.
- Rhythm : regular.
- Response to atropine : -ve (ventricular escape phenomenon).
- Respiratory sinus arrhythmia : -ve .

2- Neck vein : normal with occasional cannon A waves.

3- Auscultation : Variable S_1 with occasional sounds.

ECG :



- QRS : slow, regular & **wide abnormal** (bizarre) shaped.
- P waves : normal rate & shape.
- No fixed relation between P waves & QRS complexes (Atrioventricular dissociation)

Treatment :

- Treatment of the cause.
- Atropine.
- Artificial pacemaker : the treatment of choice.

In one word :

- ✓ **Sinus bradycardia** : is the same like **sinus tachycardia** but slow.
- ✓ **Nodal rhythm** : is the same like **Paroxysmal nodal tachycardia** but slow.
- ✓ **Complete heart block** : is the same like **ventricular tachycardia** but slow.

Atrial fibrillation

Definition :

It is a condition in which there are rapid **irregular** impulses (400-600/min) arise from the atria by multiple ectopic foci (*so the atria don't contract effectively*) & due to physiological delay at AVN , not all impulses are conducted to the ventricles.



Notice that there are multiple foci ending in ineffective atrial contraction

Etiology :

- **Mitral stenosis & thyrotoxicosis** . 🎵 🎵
- **Constrictive pericarditis & Cardiac surgery**.
- **Lone AF (idiopathic)** : especially in elderly.
- **Other causes : like scheme**.

Clinical picture :**Symptoms :**

- The same as scheme .
- **Palpitation** : rapid , irregular & may be paroxysmal or sustained.
- **Duration of the disease** : may be long .

(the patients may accommodate for a new rhythm & palpitation disappears)

- **Thromboembolism** : ineffective atrial contraction predisposes to stasis of blood and may lead to thrombosis & systemic emboli (e.g. hemiplegia)

Signs :

1- Radial pulse :

- **Rate** : usually rapid (100 – 150 /min)
may be slow as in patients on digitalis.
- **Rhythm** : ✎ marked irregularity (*you can't count 4 successive regular beats*)
✎ Pulse deficit (*apical pulse - radial pulse*) : > 10/min.
- **Response to carotid massage** : may ↓HR due to decreased AV conduction.
- **Respiratory sinus arrhythmia** : -ve .

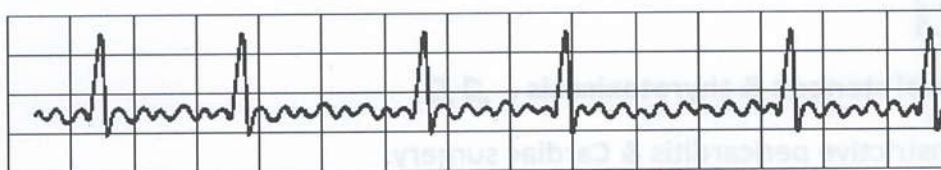
✎ If the radial pulse becomes regular & slow in a case of AF : CHB is suspected.

✎ If the radial pulse become regular & rapid in a case of AF : VT is suspected.

2- Neck vein : absent A wave.

3- Auscultation : Variable intensity of S_1 .

ECG :



- **P wave** : absent & replaced by fibrillation (F) waves .
- **QRS** : normal in shape but irregular in rhythm.

Treatment :

The acute management of AF involves **3 strategies** :

1- Reversion to normal sinus rhythm:

Methods : ✎ Electrical cardioversion.

✎ Drugs : quinidine , flacinide, propafenone or amiodarone.

Indication :

- ✗ Recent onset of AF.
- ✗ No history of recent embolism.
- ✗ No significant left atrial enlargement.

Precautions :

- ✗ Anticoagulant must be given at least 2 weeks before reversion to decrease the risk of embolization.
- ✗ Discontinuation of digitalis before electrical cardioversion is a must.

2- Control of ventricular rate : by β blocker , Ca channel blocker or Digitalis .

3- Prevention of thromboembolism : by warfarin or aspirin.

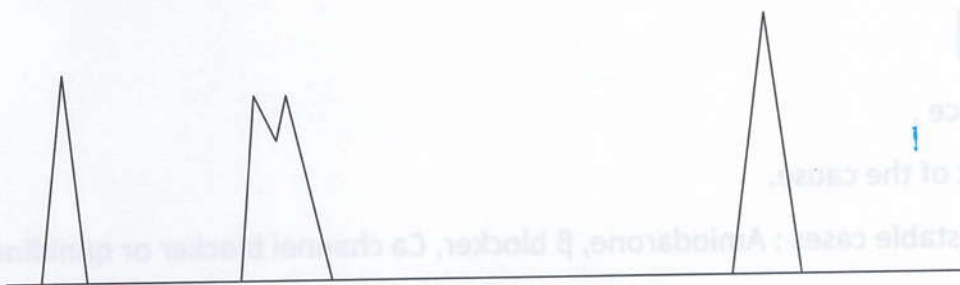
NB: - In some cases atrial fibrillation is better treated by anticoagulant therapy & control of ventricular rate without any trial to return to sinus rhythm.
 - Recurrent AF is treated by long use of propafenone, flacinide or amiodarone.

Premature beats (Extrasystoles)

Definition :

It is an ectopic impulses arising from the atria , AVN or ventricles before the expected next beat causing what is called premature beat.

- Premature beats occur during relative refractory period (RRP)



Notice that the premature beat is followed by compensatory pause & forceful contraction

Etiology :

- Physiological : Emotions , smoking or excessive coffee.
- Pathological : The same as scheme.

Clinical picture :**Symptoms :**

- Asymptomatic in most cases.
- Occasional irregular palpitation .

Signs :**1- Radial pulse :**

- **Rate** : normal , tachy or bradycardia.
- **Rhythm** : ✗ Occasional irregularity.
✗ Pulse deficit : < 10 / min.
- **Response to exercise** : The irregularity disappears due to ↓ in diastolic period.

2- Neck vein : Normal wave with occasional irregularity.

3- Auscultation : Normal sounds with occasional irregularity.

ECG :

ventricular premature beats are wide bizarre QRS not preceded by P wave & followed by compensatory pause.

Treatment :

- 1- Reassurance .
- 2- Treatment of the cause.
- 3- In chronic stable cases : Amiodarone, β blocker, Ca channel blocker or quinidine.
- 4- Lidocaine (IV) in emergency cases.

Wolf-Parkinson-White (WPW) syndrome :

- It is accessory pathway that connects the atrium & ventricle & can bypass the AVN.
- So, AF is a very serious arrhythmia in these patients, it may lead to ventricular fibrillation.
- WPW is associated with thyrotoxicosis, mitral valve prolapse, HCM & more common in men.
- **Treatment :** Amiodarone , β blocker . Radiofrequency ablation is the treatment of choice.
- Digitalis & verapamil should be avoided ($\uparrow$ conduction through the accessory pathway).

Treatment of arrhythmias**I- Pharmacological (Antiarrhythmic drugs) :**

CLASS	DRUGS	MAIN USES
Class I : Na channel blockers (slows the depolarization)		
Class IA	Quinidine , Procainamide.	Broad spectrum.
Class IB	Lidocaine , Phenytoin.	Ventricular arrhythmias.
Class IC	Flacainide , Propafenone.	Broad spectrum.
Class II : β blockers	Propranolol , Atenolol , Esmolol	Tachyarrhythmias. Premature beats.
Class III : K channel blockers	Amiodarone , Bretylium.	Broad spectrum.
Class IV : Ca channel blockers	Verapamil , Diltiazem.	Atrial tachyarrhythmias.
Others :	Adenosine ($\downarrow$ automaticity & conductivity) Digitalis ($\downarrow$ automaticity & conductivity)	PSVT Atrial tachyarrhythmias

Side effects of antiarrhythmic drugs :

- **Proarrhythmias :** new arrhythmias induced by the drug .

➤ **Quinidine :**

- ✗ Allergy & hypotension .
- ✗ Cinchonism (headache, vomiting , tinnitus & blurring of vision).
- ✗ Digitalis toxicity .

➤ **Lidocaine :** 3M

- ✗ Mental confusion.
- ✗ Myocardial depression.
- ✗ Muscle twitching.

➤ **Amiodarone :** due to its tendency to accumulate in body tissue it may lead to:

- ✗ CNS : Dizziness, depression , tremors.
- ✗ Corneal deposits.
- ✗ Thyroid dysfunctions (hyper or hypo thyroidism)
- ✗ Pulmonary fibrosis.
- ✗ Elevation of hepatic enzymes.
- ✗ Constipation.
- ✗ Skin pigmentation.

II- Non pharmacological :

- DC cardioversion .
- Implantable cardioverter defibrillator (ICD).
- Radiofrequency catheter ablation .
- Artificial pacemaker .(temporary , permanent).

I have not failed. I've just found 10,000 ways that won't work.

Thomas Edison

Cardiac arrest

Definition :

Sudden & complete loss of cardiac function. It is usually diagnosed clinically by the absence of a pulse with diminished responsiveness. Without immediate intervention it will almost always lead to death.

Causes :

70% of sudden cases have been attributed to coronary heart disease.

I. Shockable : Those responsive to defibrillation

a) Ventricular fibrillation :

- This is the most common cause of cardiac arrest.
- Rapid irregular uncoordinated contractions with no cardiac output.
- It can cause irreversible brain damage after 4 minutes.

b) Pulseless ventricular tachycardia : wide complex regular tachycardia associated with no cardiac output.

II. Non-shockable (Non VF/VT) : Those unresponsive to defibrillation

- The prognosis of this group is much less favorable than with VF/VT
- Defibrillation is NOT indicated.

a) Ventricular asystole :

- Here, there is no electrical activity of the ventricles.
- It is due to failure of conducting system of the heart, or massive myocardial infarction.

b) Pulseless electrical activity : (Electromechanical dissociation)

- Here, there is no effective cardiac output despite the presence of normal electrical activity. (Rhythm on monitor, without detectable pulse)
- This may be caused by : Tension pneumothorax, Massive pulmonary embolism, Myocardial rupture.

Precipitating factors of cardiac arrest :

5 H & 5 T

5 Hs:

- Hypovolemia
- Hypoxia.
- Hydrogen ions (acidosis)
- Hyperkalemia & Hypokalemia.(Hyper more common)
- Hypothermia.

5 Ts:

- Thrombosis (coronary/pulmonary).
- Toxins : CO , Cyanide.
- Tamponade. (I mean cardiac tamponade)
- Tension pneumothorax.
- Traumatic cardiac arrest.

Management of cardiac arrest : (Cardiopulmonary resuscitation, CPR)

- I. Basic life support (BLS)
- II. Advanced cardiac life support (ACLS)

I. Adult basic life support (ABLS) :

- Aim : is to maintain circulation until more definitive treatment with advanced life support can be given.
- BLS has 3 components : the ABCs of life support (ABC or **CAB**)

1. Airway : Open the airway

- Head tilt (if no spine injury) + chin lift & maintain clean and clear airways.
- Tracheal intubation (which is considered a component of advanced life support) is the preferred method of maintaining a patent airway in unconscious patients with cardiac arrest.
- Prior to intubation, an oropharyngeal airway (an S-shaped device passed over the tongue and into the pharynx) can be used to keep the flaccid tongue from falling back and occluding the oropharynx.

2. Breathing : Provide positive pressure ventilations

- Use specialized bag and mask system. Otherwise, mouth-to-mouth breathing.
- Avoid hyperventilation : the problem here is the increase in positive intrathoracic pressure which may lead to decrease of both ventricular filling & coronary perfusion.
- To minimize the adverse effects of hyperventilation, avoid using lung inflation rates above 10 breaths/min.

3. Circulation : (Chest compressions or a cardiac thump)

- Give 15 compressions to 2 breaths (15 : 2).
- Performed by placing the heel of the dominant hand on the lower half of the sternum, with the non dominant hand on top. The both arms are kept straight.
- The sternum should be depressed 5cm inward, and should be allowed to recoil completely before the next compression.
- The recommended rate of compression : at least 100/min

II. Advanced cardiac life support : ACLS

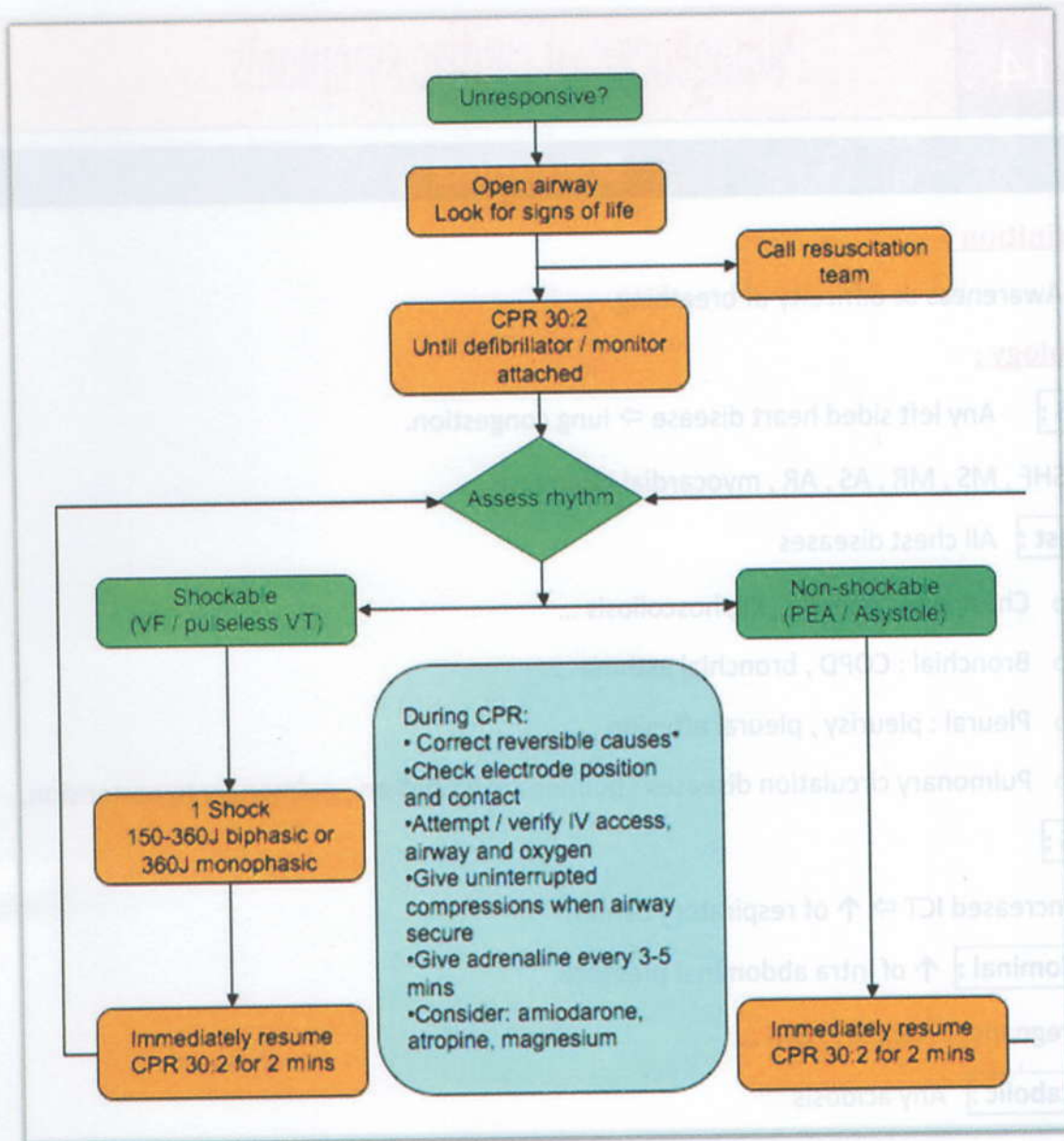
- ACLS includes a verities of more specific interventions e.g. airway intubation, mechanical ventilation, defibrillation and drugs given during cardiac arrest.
- The top priority in ACLS is to rapidly assess & treat the abnormal cardiac rhythm by attaching defibrillator/monitor.

a) Defibrillation :

- Direct current (DC) cardioversion is the single most effective resuscitative measure for improving survival in shockable cardiac arrest (VF/VT).
- The recommended energy level for the first shock is 200 joules for biphasic shocks and 360 joules for monophasic shocks.
- If the first shock is ineffective, two additional shocks can be done (don't forget to perform CPR between successive shocks).

b) Drugs administration during CPR :

- ✍ Vasopressors :
 - Epinephrine (1mg IV, repeated every 5 min): used in all cardiac arrests.
 - Vasopressin : 40 units IV as a single dose.
- ✍ Dopamine : used in cases with cardiogenic shock.
- ✍ Atropine (1mg frst dose, repeated every 5 min) : used in cases with bradycardia, or as an adjunct to vasopressors for asystole & PEA.
- ✍ Antiarrhythmic drugs : according to the type of arrhythmia
 - Amiodarone : 300mg IV
 - Lidocaine : 1mg/kg IV
- ✍ Ca : It is not indicated for routine use in cardiac arrest. It is indicated in severe hypocalcemia, Ca channel blocker toxicity, cardiac arrest after multiple transfusion with citrated blood.



Post-resuscitation care :

- Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury.
- Strict glycemic control : Avoid hyper or hypoglycemia .
- Therapeutic hypothermia : Mild hypothermia is neuroprotective and improves outcome. The target body temperature is 32C to 34C for no less than 12 hours and no longer than 24 hours.

14

Pathogenesis of cardiac symptoms

Dyspnea

Definition :

Awareness or difficulty of breathing.

Etiology :

CVS : Any left sided heart disease $\Rightarrow$ lung congestion.

LSHF , MS , MR , AS , AR , myocardial infarction

Chest : All chest diseases

- Chest wall : obesity , Kyphoscoliosis ...
- Bronchial : COPD , bronchial asthma
- Pleural : pleurisy , pleural effusion
- Pulmonary circulation diseases : pulmonary embolism , pulmonary hypertension.

CNS :

Increased ICT $\Rightarrow$ $\uparrow$ of respiratory center.

Abdominal : $\uparrow$ of intra abdominal pressure.

Pregnancy , Ascites , HSM

Metabolic : Any acidosis

DKA , Lactic acidosis , Uremic acidosis ...

Pathogenesis of cardiac dyspnea : 7

- 1- Decrease in the vital capacity due to increase the amount of blood present in the lungs.
- 2- Exaggeration of Hering-Breuer reflex due to rigid alveoli.
- 3- Churchill-Cope reflex : It occurs in pulmonary congestion which leads to stimulation of respiratory center.
- 4- Fatigue of respiratory muscle due to LCOP.

5- Pulmonary edema $\Rightarrow$ non functioning alveoli.

6- Pleural & pericardial effusion lead to mechanical compression of the lungs.

7- Hypoxia $\Rightarrow$ stimulation of respiratory center.

Grades of dyspnea : New York Heart Association (NYHA)

- Grade I : Dyspnea on extra ordinary effort.
- Grade II : Dyspnea on ordinary effort.
- Grade III : Dyspnea on less than ordinary effort .
- Grade IV : Dyspnea even at rest.

Orthopnea :

- Dyspnea on lying flat , partially relieved by sitting.

- This occurs due to :

- Increased venous return which increases the lung congestion.
- Elevation of diaphragm.

- No one can blame me when I say that orthopnea is not specific to cardiac diseases , It also may occur due to chest or abdominal causes.

Paroxysmal Nocturnal Dyspnea (PND) :

- Attacks of dyspnea & cough with frothy expectoration occurring during night 1 – 2 hours after sleep & after a few minutes the patient feels better & goes back to sleep.
- If the condition is accompanied with chest wheezes , it is called cardiac asthma.
- Cardiac asthma is a state of impending pulmonary edema.
- PND is a specific symptom of left sided heart lesion especially LSHF.
- Pathogenesis of PND : During sleep , there are :
 - i- Vagal predominance $\Rightarrow$ causes further weakness of the contraction of left ventricle.
 - ii- Absorption of edema fluid into the circulation $\Rightarrow \uparrow$ VR $\Rightarrow \uparrow$ pulmonary congestion.

- **The condition must be differentiated from bronchial asthma.**

	Cardiac asthma	Bronchial asthma
Age	Usually old	Usually young
Duration of the attack	Usually short	Usually long
Time of the attack	1-2 hours after sleep	Early morning
Frequency	Low frequency	More frequent
Dyspnea	Mainly inspiratory	Mainly expiratory
Expectoration	Frothy & may blood tinged sputum.	Thick sputum
Cardiac examination	Murmurs & gallop	Normal
Chest examination	Crepitation > rhonchi	Rhonchi > crepitation
Circulation time (arm to tongue)	Prolonged	Normal
Morphine	Improvement	contraindicated

Hemoptysis

Hemoptysis in a cardiac patient may be due to : 7

1. **Pulmonary apoplexy** : frank severe hemoptysis occurring mainly in **MS** due to rupture of bronchial varices.
2. Chest infection due to lung congestion.
3. Acute pulmonary edema : blood tinged sputum.
4. Pulmonary infarction.
5. Myocardial infarction : Dressler syndrome (*post infarction syndrome*).
6. Ruptured aortic aneurysm.
7. Severe systemic hypertension.

Acute pulmonary edema

Etiology :

A) **Cardiogenic pulmonary edema :**

- Acute left sided heart failure e.g. myocardial infarction.
- On top of chronic LSHF : MS with aggravating factor as AF.

B) **Non cardiogenic pulmonary edema :** (Acute Respiratory Distress Syndrome – ARDS)

Diffuse alveolar damage (DAD) characterized by $\uparrow$ capillary permeability , pulmonary edema & refractory hypoxia , caused by :

- Sepsis .
- Aspiration of gastric contents.
- 2 P : pneumonia , pancreatitis.
- 2 B : Burn , Blood transfusion.
- Terminal renal & hepatic failure.

Clinical picture of acute pulmonary edema :

- Severe dyspnea at rest & orthopnea.
- Sense of impending death.
- Sweating & irritability.
- Cyanosis. ➤ Crepitation.
- Cough with frothy pink sputum.
- Features of the Cause :MI.

Differential Diagnosis : DD of acute dyspnea : MI & 6 P see p 104

Investigation :

- Investigations of heart failure.
- Investigations for the cause e.g. MI : ECG & cardiac enzymes.
- Arterial blood gases : $\downarrow$ O_2 , $\downarrow$ PCO_2 ($PCO_2 \uparrow$ in late cases)
- Investigations for ARDS : No LSHF (PCWP < 18 mmHg , EF : normal)

Treatment : see p 15

Syncope

Definition :

Sudden, transient loss of consciousness due to cerebral ischemia followed by spontaneous recovery.

Causes :

1- **Vasomotor syncope :**

- i- Vaso vagal attack : bad sight , bad smell , exposure to fear.....
- ii- Carotid sinus syndrome : shaving , carotid sinus massage.

2- **Orthostatic syncope :** (postural hypotension)

- **A**utonomic neuropathy as in DM.
- **A**disson's disease.
- **A**ntihypertensive drugs : α blockers , α methyl dopa .
- Weakness of the muscles of the lower limbs.

3- **Cardiac :**

- A) Exertional : AS , LSHF .
- B) At rest : CHB , VT.
- C) Positional (syncope in certain position) : Left atrial myxoma.
- D) Acute decrease of VR e.g.hemorrhage.
- E) Hypoxia : as in F₄ (Cyanotic spell)

4- **Cerebral :**

- Transient ischemic attack (TIA).
- Hypertensive encephalopathy.
- Cerebral embolism.

5- **Situational :**

Cough syncope :

Severe cough $\Rightarrow$ $\uparrow$ intrathoracic pressure $\Rightarrow$ $\downarrow$ VR $\Rightarrow$ $\downarrow$ cerebral blood flow $\Rightarrow$ syncope.

Micturation syncope : occurs in old men with BPH.

Shock

Definition: Medically, shock is defined as **inadequate tissue perfusion**.

Types & Causes: there are 4 types:

1- **Hypovolemic shock:** (*the most common*)

It is due to volume loss e.g.

- Blood loss : hemorrhage.
- Plasma loss : burn.
- Fluid loss : vomiting, diarrhea, dehydration (DKA)

2- **Cardiogenic shock:** Pump failure (↓ *contractility*)

- Extensive MI (> 40 % of LV mass)
- Arrhythmias.
- Acute MR .

3- **Obstructive shock.** Mechanical obstruction to COP (*extrinsic cardiogenic*)

- Cardiac tamponade
- Tension pneumothorax
- Massive pulmonary embolism.

4- **Distributive shock:** systemic vasodilatation ⇔ ↓ peripheral vascular resistance

- Septic shock : sepsis with hypotension.
- Anaphylactic shock : caused by a hypersensitivity reaction to an allergen.
- Neurogenic shock : Failure of the nervous system to control diameter of blood vessels.
- Adrenal insufficiency.

Pathphysiology: (all types of shock)

- Reduction of tissue perfusion.
- Damage of cellular membranes.
- Leakage of lysosomal enzymes.
- Reduction of cellular energy stores.
- Cell death.

Hemodynamic changes in shock :

	CO	SVR	CVP
Hypovolemic	↓	↑	↓
Cardiogenic	↓	↑	↑
Obstructive	↓	↑	↑
Distributive	Septic ↑, Neurogenic ↓	↓	↓

- Tissue perfusion is driven by blood pressure : $\text{Blood Pressure} = \text{CO} \times \text{SVR}$

- COP , in turn depends on the venous return.

- CVP or PCWP are used as index of venous return.

So, these variables can be used to describe types of shock :

- **CO** (Cardiac output) ↓ in all types except in a case of Septic shock.
- **SVR** (systemic vascular resistance) : ↑ in all types (due to VC) except in distributive shock (VD)
- **CVP** (Central venous pressure) : pressure in SVC , measured by pulmonary (Swan Ganz) Catheter.
- **PCWP** (Pulmonary Capillary Wedge Pressure) : It's a reflection of LA pressure (by pulmonary catheter)

Clinical picture:➤ **General manifestations common to all types of shock :**

1. Hypotension (systolic < 100 mmHg , mean BP < 60 mmHg)
2. Tachycardia > 100/m (↑ sympathetic) , except in Neurogenic shock.
3. Tachypnea. (due to metabolic acidosis & pulmonary congestion)
4. Oliguria (< 30 ml/h).
5. Drowsiness , confusion .
6. Cold extremities except in early distributive shock.
7. Multi organ failure (MOF).

➤ **Specific manifestations :**1- **Hypovolemic shock :**

- The same as general manifestations.
- Clinical picture depends on volume lost.

It is classified according to the percentage of blood loss into 4 classes :

(The same as the score in a tennis game : 15 - 30 - 40 - GAME OVER)

- Class I : up to 15 %
- Class II : 15 - 30 %
- Class III : 30 - 40 %
- Class IV : > 40 %

Cardiogenic shock :

- The same as general manifestations.
- Clinical picture of the cause e.g. MI : chest pain.
- Clinical manifestations of acute pulmonary edema : dyspnea , crepitations...
- Congested neck vein.

Obstructive shock :

- The same as general manifestations.
- C/P of the cause e.g. Cardiac tamponade (↑JVP, ↓BP , distant heart sounds)

Distributive shock :

Septic shock :

- The same as general manifestations.
- Evidence of fever & localized infection.
- Warm flushed extremities (warm shock)
- Strong pulse.

Anaphylactic shock :

- Warm flushed extremities.
- Erythema , Urticaria , angio edema , bronchospasm.

Neurogenic shock : Hypotension with bradycardia.

Investigations :**Laboratory :**

- Blood picture.
- Blood culture.
- Liver & renal function tests.
- Blood gases.
- Blood glucose.
- Lactic acid level.

Imaging :

- X ray , ECG , Echo : for cardiac causes of shock.
- US & CT abdomen : for abdominal infections & intraperitoneal hemorrhage .

Treatment :

Goal : to restore normal tissue perfusion.

General measures :

- Monitoring of hemodynamics : CVP , CO , SVR.
- Trendelenburg position . (legs up , head down)
- Patent airway.
- Oxygen therapy.
- IV fluid therapy in noncardiogenic shock patients.

Specific treatment : according to the type of shock.

Hypovolemic shock :

- Identify source of volume depletion.
- Blood transfusion in a cases of bleeding.

Cardiogenic shock :

- Treatment of the cause e.g. MI
- Dopamine , Dobutamine .
- Mechanical assist devices : Intra aortic balloon counterpulsation.

Obstructive shock: *relief of the obstruction is life saving*

- If cardiac tamponade is present, urgent pericardiocentesis is essential.
- Tension pneumothorax must be treated with needle thoracostomy.
- Massive pulmonary embolism requires urgent thrombolysis or surgical removal.

Anaphylactic shock :

- Adrenaline.
- Hydrocortisone IV
- Antihistaminic.

Septic shock :

- Treatment of infection by antibiotics.
- Vasopressor as Adrenaline.

15

Heart in systemic diseases

Neurologic disorders and heart diseases :

1. Heridofamilial neuro-myopathic disorders : e.g. Duchenne myopathy

- Dilated cardiomyopathy.
- Arrhythmias e.g. sinus tachycardia, VT, RBBB.

2. Friedreich's ataxia.

- Hypertrophic cardiomyopathy. (more common)
- Dilated cardiomyopathy. (less common)

3. Myotonia muscle dystrophy.

- Myocardial dystrophy : HF.
- Arrhythmias & heart block.

4. Cardiovascular abnormality of acute cerebral injury :

- Arrhythmias.
- Cardiopulmonary arrest.
- Myocardial stress injury (catecholamine storm)
- Repolarization abnormalities : depressed ST segment, inverted T wave.

Cardiac manifestations of renal failure :

1. HF.
2. Systemic hypertension in 80% of patients with CRF.
3. Hypertrophic cardiomyopathy.
4. Arrhythmias : due to electrolytes disturbance.
5. Painless pericarditis.
6. Accelerated coronary atherosclerosis.
7. Cardiac calcification → Valvular heart disease.
8. Cardiovascular complications during dialysis.

The Heart & endocrinal diseases :

Acromegaly :

1. Cardiomegaly.
2. Hypertension (up to 50% of cases)
3. Atherosclerosis : due to hypertension, abnormal lipid & carbohydrate metabolism.
4. Heart failure : in 20% of cases.

Thyrotoxicosis : see endocrinology book

Myxedema :

1. Pericardial effusion.
2. Hypertension.
3. Atherosclerosis due to hypercholesterolemia.
4. ECG changes :
 - Sinus bradycardia.
 - Low voltage.
 - Heart block.

Cushing syndrome :

1. Hypertension.
2. CHF.
3. Stroke.
4. MI.
5. Accelerated atherosclerosis due to hyperlipidemia.

Diabetes mellitus :

1. **Ischemic heart disease :** (Angina & MI)
 - May be silent.
 - Higher mortality.
2. **Hypertension :** Causes of hypertension in diabetics : see endocrinology book.
3. **Diabetic cardiomyopathy.**
4. **Cardiac autonomic neuropathy :**
 - Arrhythmia.
 - Fixed HR.
 - Postural hypotension.
 - Silent MI.
5. **Vascular dysfunction in diabetes :**
 - Microvascular : diabetic retinopathy, nephropathy & neuropathy.
 - Macrovascular (atherosclerosis) : coronary, cerebral, renal, peripheral.

If you can't explain it simply, you don't understand it well enough.

Albert Einstein

Cardiac parameters

- Heart rate (*HR*) : 60 - 90 beats / min.
- Cardiac output (*CO*) : 4 - 6 L / min.
- Stroke volume (*SV*) : 60 - 100 ml / beat.
- Right atrial pressure (*RAP*) : 0 - 6 mmHg.
- Central venous pressure (*CVP*) : 0 - 6 mmHg.
- Left atrial pressure (*LAP*) : 6 - 12 mmHg.
- Pulmonary capillary wedge pressure (*PCWP*) : 6 - 12 mmHg.
- Right ventricular pressure (*RVP*) :
 - Systolic : 15 - 30 mmHg.
 - Diastolic : 0 - 8 mmHg.
- Right ventricular End Diastolic volume (*RVEDV*) : 100 - 160 ml.
- Right ventricular Ejection fraction (*RVEF*) : 40 - 60 %
- Pulmonary artery pressure :
 - Systolic : 15 - 30 mmHg.
 - Diastolic : 8 - 15 mmHg.
 - Mean : 10 - 20 mmHg.
- Left ventricular pressure (*LVP*) :
 - Systolic : 90 - 140 mmHg.
 - Diastolic : 4 - 12 mmHg.
- Arterial blood pressure :
 - Systolic (*SBP*) : 90 - 140 mmHg.
 - Diastolic (*DBP*) : 60 - 90 mmHg.
 - Mean (*MAP*) : 70 - 105 mmHg.

MCQ

1- The Cause of silent myocardial infarction may be :

- a) Athletes
- b) Obese
- c) Females
- d) Diabetes mellitus

2- Which of the following best describes the effect of calcium ions on the myocardium?

- a) Positively inotropic
- b) Negatively inotropic
- c) Positively chronotropic
- d) Negatively chronotropic

3- Wide fixed splitting of the second heart sound occur in :

- a) Mitral stenosis
- b) Mitral regurge
- c) Atrial septal defect
- d) Ventricular septal defect

4- In the absence of critical stenosis of the coronary arteries, angina pectoris occurs most frequently with which of the following valvular heart diseases?

- a) Mitral stenosis
- b) Mitral regurge
- c) Aortic stenosis
- d) Aortic regurge

5- Which of the following cardiac lesion has the highest risk of developing infective endocarditis?

- a) VSD
 - b) ASD
 - c) Mitral valve prolapse with regurgitation
 - d) MS
- VSD : high risk lesion for IE
 - MS : intermediate risk
 - ASD : Low risk

6- Which lipid pattern suggests the lowest risk for CAD?

- a) Total cholesterol 215 mg /dL, HDL cholesterol 28 mg/dL
- b) Total cholesterol 215 mg /dL, HDL cholesterol 43 mg/dL
- c) Total cholesterol 180 mg /dL, HDL cholesterol 29 mg/dL
- d) Total cholesterol 202 mg /dL, HDL cholesterol 45 mg/dL

This combination has the lowest risk, although the total cholesterol is on borderline, the high HDL cholesterol is protective.

- Total cholesterol : N → <200, borderline : 200 – 240, high ≥ 240
- HDL : N → 40-50 in ♂ 50-60 in ♀
- LDL : < 70 ideal for people at very high risk of heart disease, < 100 ideal for people at high risk of heart disease.

7- Which one of the following is characteristic of atrial myxoma?

- a) It usually originate in the right atrium
- b) The clinical signs can mimic severe mitral regurgitation
- c) Recurrence is frequent, even after successful surgical removal
- d) Echocardiogram is diagnostic in most cases

75% of cases originate in left atrium, the clinical signs mimic MS

8- A 52-year-old woman with no prior medical history presents in the emergency department with a 3-hour episode of crushing substernal chest pain. The pain radiates to her arm and neck. An ECG reveals ST segment elevation in leads II, III and aVF. The patient has no obvious contraindication to anticoagulation. Which of the following is the most optimal treatment at this time?

- a) Administration of IV fluids
- b) Administration of aspirin and heparin only
- c) Administration of thrombolytic therapy, heparin, and aspirin
- d) Cardiac surgery to bypass the occluded vessel

NB : if the patient is hypotensive, IV fluids may be needed.

9- The most effective diagnostic modality in a case of subacute bacterial endocarditis is :

- a) ECG
- b) Transthoracic echocardiogram (TTE)
- c) Transesophageal echocardiogram (TEE)
- d) Cardiac catheterization

10- Which of the following therapies has been shown to increase survival in a case of post myocardial infarction patients who have ejection fraction > 50%?

- a) Angiotensin-Converting enzyme inhibitor
 - b) Beta blocker
 - c) Digoxin
 - d) Loop diuretic
- Beta blockers have been shown to improve survival after myocardial infarction (MI) by decreasing both oxygen demand and the incidence of ventricular arrhythmia.
 - Angiotensin-converting enzyme (ACE) inhibitors (choice A), such as enalapril, have been shown to improve survival in post-MI patients who have ejection fractions less than 40%.

11- A 51-year –old man is brought to the emergency department for chest pain. The patient has chronic stable angina that is usually precipitated by activity and relived by rest. About 3 weeks ago, his physician prescribed sildenafil (Viagra), and he has been using the drug with success. This morning, he developed acute onset of substernal chest pain, radiating to his left arm. This pain is not relived by rest. The patient last took a sildenafil the night before. Which of the following treatments is absolutely contraindicated in this situation?

- a) Captopril
- b) Metoprolol
- c) Nitroglycerin
- d) Tissue plasminogen activator (tPA)

The co-administration of nitrates within 24 hours after taking sildenafil is absolutely contraindicated.

The vasodilatory effects of nitrates are amplified when administered in the presence of sildenafil (Viagra), which can lead to refractory and life-threatening hypotension. Therefore, patients using sildenafil should be instructed to report their use on presentation to any emergency department and to never take nitrates while using the drug.

12- A 57-year-old man presents to his physician for follow-up. He has a positive family history for coronary artery diseases and he has smoked one-half pack of cigarettes per day for the past 20 years. Which of the following lipid patterns would most strongly suggest the need for pharmacologic therapy in this patient?

- a) Total cholesterol 180 mg/dL, LDL cholesterol 140 mg/dL
- b) Total cholesterol 230 mg/dL, LDL cholesterol 100 mg/dL
- c) Total cholesterol 245 mg/dL, LDL cholesterol 165 mg/dL
- d) Total cholesterol 285 mg/dL, LDL cholesterol 100 mg/dL

- A patient with 2+ risk factors and an LDL of geater than 160mg/dL nedds medical therapy.
- A total cholesterol of 180 mg/dL, LDL cholesterol of 140 mg/dL (choice a) : in this patient could be managed with a trial of dietary modification and education.
- A total cholesterol of 285 mg/dL with an LDL cholesterol of 100 mg/dL (choice d) : does not require drug therapy. The total cholesterol is elevated, but the LDL Is not, suggesting high HDL level.

13- In a patient with central chest pain at rest :

- a) Relief of pain by nitrates excludes an esophageal cause
- b) Features of autonomic disturbance are specific to cardiac pain
- c) Intrascapular radiation suggests the possibility of aortic dissection
- d) Myocardial ischemia radiates to the neck but not the jaw

Notice that autonomic disturbance may occur in severe pain from any cause.

14- Recognised causes of secondary hypertension include EXCEPT

- a) Persistent ductus arteriosus
- b) Coarctation of the aorta
- c) Primary hyperaldosteronism
- d) Acromegaly

15- As regarded to the auscultatory findings listed below, which is correct?

- a) Third heart sound-opening of mitral valve
- b) Varying intensity of first heart sound-atrioventricular dissociation
- c) Soft first heart sound-mitral stenosis
- d) Fourth heart sound-atrial fibrillation

16- In the investigation of suspected angina pectoris :

- a) Physical examination is of no clinical value
- b) The resting ECG is usually normal
- c) Exercise-induced elevation in blood pressure indicates significant ischemia
- d) A normal ECG during exercise excludes angina pectoris

Physical examination is important to exclude anemia and valvular stenosis.

17- In the treatment of patients with angina pectoris :

- a) Aspirin reduces the frequency of angina attacks.
- b) Glyceryl trinitrate is equally effective when swallowed as when taken sublingually
- c) Calcium antagonists may cause peripheral edema
- d) β blockers are more effective than other ant-anginal agents

18- In the treatment of acute myocardial infarction :

- a) Aspirin given within 6 hours of onset reduces the mortality
- b) Diamorphine is better given intramuscular than by any other route
- c) Immediate calcium channel blocker therapy reduces the early mortality rate
- d) Nitrate therapy reduces the early mortality rate

19- Drug therapies which improve the long-term prognosis after myocardial infarction include EXCEPT :

- a) Aspirin
- b) Calcium antagonists
- c) ACE inhibitors
- d) β -blockers

20- Clinical features suggesting aortic stenosis include :

- a) Late systolic ejection click
- b) Slapping apex beat
- c) Syncope associated with angina
- d) Loud second heart sound

21- Disorders associated with aortic regurgitation include EXCEPT

- a) Ankylosing spondylitis
- b) Marfan's syndrome
- c) Syphilitic aortitis
- d) Persistent ductus arteriosus

22- In atrial septal defect :

- a) The lesion is usually of primum type
- b) The initial shunt is right to left
- c) Splitting of the second heart sound increases in expiration
- d) The ECG typically shows right bundle branch block

23- Dilated (congestive) cardiomyopathy is EXCEPT

- a) Usually idiopathic
- b) Associated with specific ECG changes
- c) Associated with chronic alcohol misuse
- d) Caused by Coxsackie A, influenza and HIV infection

24- All of the following are recognized complications of acute MI EXCEPT

- a) Acute mitral regurge
- b) Acute aortic regurge
- c) Dressler's syndrome
- d) Left ventricular aneurysm

25- Which of the following tests is most sensitive and specific for the diagnosis of coronary artery disease?

- a) Stress ECG
- b) Stress echocardiography
- c) Cardiac catheterization and coronary angiography
- d) Multi slice CT

26- Mitral stenosis...

- a) Graham steel murmur may occur
- b) Causes enlargement of both left atrium & left ventricle
- c) May lead to development of non cardiogenic pulmonary edema
- d) Rarely causes AF

Graham steel murmur is an early diastolic murmur heard in the second intercostal space to the left of the sternum. It is associated with pulmonary valve regurgitation in pulmonary hypertension.

27- Sudden death may occur in

- a) AS
- b) ASD
- c) PDA
- d) Constrictive pericarditis

28- Clinically, severity of MS is best assessed by

- a) Diastolic shock
- b) Proximity of S2-opening snap gap
- c) Paroxysmal nocturnal dyspnea
- d) Shorter duration of mid diastolic murmur

29- Clubbing is not a feature of :

- a) Fallot's tetralogy
- b) Left atrial myxoma
- c) Right to left shunt
- d) Acute bacterial endocarditis

30- Digitalis toxicity is associated with all except

- a) Wenckebach block
- b) Ventricular bigeminy
- c) Paroxysmal atrial tachycardia with block
- d) Mobitz type II block

31- Hemoptysis may be found in

- a) Left ventricular failure
- b) Right ventricular failure
- c) Pulmonary stenosis
- d) Left to right shunt

32- Which is not a major manifestation of Jones criteria in rheumatic fever

- a) Chorea
- b) Erythema nodosum
- c) Subcutaneous nodule
- d) Polyarthrititis

NB : Erythema marginatum not nodosum ☺

33- Atrial myxoma may be associated with all except

- a) Fever
- b) Splenomegaly
- c) Clubbing
- d) High ESR

34- S4 is not associated with

- a) AS
- b) Hypertrophic cardiomyopathy
- c) Chronic mitral regurge
- d) Systemic hypertension

35- Incidence of infective endocarditis is least in

- a) MI
- b) ASD
- c) PDA
- d) VSD

36- Which enzyme rise earliest in acute myocardial infarction

- a) AST
- b) ALT
- c) CPK
- d) LDH

37- Which of the following β blockers is used in heart failure

- a) Carvedilol
- b) Atenolol
- c) Labetalol
- d) Pindolol

38- Drug of choice in acute management of PSVT is

- a) Amiodarone
- b) Verapamil
- c) Metoprolol
- d) Adenosine

39- Earliest valvular lesion in acute rheumatic carditis is

- a) MS
- b) AI
- c) MI
- d) AS

40- Commonest heart valve abnormality reveled after acute myocardial infarction is

- a) AI
- b) MI
- c) AS
- d) AI

41- Which one of the following is the most reliable feature of chest pain secondary to ischemic heart disease?

- a) Retrosternal location
- b) Radiation to the lower jaw
- c) ECG shows T wave inversion in lead III
- d) Pain aggravated by heavy meals

- Retrosternal chest pain, though characteristic of ischemic heart disease, is also may occur in other condition such as esophageal spasm.
- Radiation to the lower jaw is the most specific characteristic of angina pain.

42- In cardiopulmonary resuscitation which of the following is true?

- a) External cardiac massage should be performed at 30 compressions/min
- b) Most patients in ventricular fibrillation will respond to 200 j shock
- c) Adrenaline should not be used in ventricular fibrillation
- d) Calcium chloride should be given

- External cardiac massage should be done at a rate of 60-80/min
- Calcium chloride is ineffective except in Ca antagonist overdose & after cardiac surgery

43- Which of the following statements is true regarding infective endocarditis?

- a) Can occur on previously normal heart valves
 - b) Can be caused only by bacteria
 - c) Streptococcus fecalis is the commonest pathogen
 - d) Occurs commonly in calcific mitral stenosis
- IE may occur in previously normal heart as occurs in IV drug addicts
- It can be caused by bacteria as well as fungi & rickettsia

44- Treatment with thiazide diuretic :

- a) Improves glucose tolerance
- b) Reduces potassium loss
- c) May precipitate gout
- d) Reduces renin level

45- Accentuation during inspiration characterizes the murmur of :

- a) AR
- b) AS
- c) TR
- d) VSD

46- All of the following antiarrhythmic drugs can not be used with heart failure except

- a) Verapamil
- b) Disopyramide
- c) Quinidine
- d) Amiodarone

Most antiarrhythmic drugs have a negative inotropic effect except amiodarone

47- Right ventricular hypertrophy may be present in all of the following conditions except

- a) Cor pulmonale
- b) ASD
- c) TS
- d) MS

48- Which of the following is absolute contraindication to thrombolysis for an acute myocardial infarction?

- a) Menstruation finished 2 days previously
- b) Proliferative diabetic retinopathy
- c) Ischemic stroke 18 months previously
- d) Total hip replacement 7 days previously

Notice that proliferative diabetic retinopathy is not an absolute contraindication, but a "relative" one.

49- All of the following are associated with increased rates of myocardial infarction except

- a) Hemochromatosis
- b) SLE
- c) Antiphospholipid syndrome
- d) Kawasaki disease

Hemochromatosis can result in a cardiomyopathy, but not MI

50- Cardiac tamponade may occur with all except

- a) Tuberculosis
- b) Rheumatic fever
- c) Rheumatoid arthritis
- d) Uremia

51- Coarctation of the aorta

- a) Is more common in women
- b) Is associated with rib notching all 12 ribs on the left
- c) Rarely causes problems in pediatric life
- d) Is associated with cerebral aneurysm

- Notching of ribs 3-8 is seen

- Coarctation can cause heart failure in the neonate

- stroke may result from hypertension or from associated berry aneurysm

52- Chest pain most commonly occurs in

- a) MS
- b) AI
- c) AS
- d) TI

53- Digoxin toxicity is more likely with

- a) Hyperkalemia
- b) Hypocalcemia
- c) Hypomagnesemia
- d) Spironolactone

Hypokalemia, hypomagnesemia & hypercalcemia worsen digitalis toxicity

54- Which of the following factors is most suggestive of a diagnosis of aortic dissection?

- a) Profound vomiting prior to pain
- b) History of syphilis
- c) Down's syndrome
- d) Hypotension

Dissection of the aorta is associated with hypertension, cocaine abuse, trauma & syphilis.

By association with coarctation, it is also associated with Turner's, but not with Down's syndrome.

55- Ventricular septal defect is associated with

- a) Pansystolic murmur once Eisenmenger's syndrome has occurred
- b) Lithium exposure in utero
- c) Aortic regurgite
- d) Heart failure on the first day of life

- The murmur disappears with the onset of Eisenmenger's syndrome as pressures equalize.

- Sub-aortic VSD is associated with AR

-Lithium exposure during development is associated with Ebstein's anomaly (Tricuspid regurgite)

56- What is the most appropriate initial management of propranolol toxicity?

- a) IV atropine
- b) IV adrenaline
- c) IV glucagon
- d) IV ciprofloxacin

Glucagon is the drug of choice for beta-blocker toxicity. It stimulates production of cAMP through non-adrenergic pathways, resulting in enhanced myocardial contractility, heart rate, and atrio-ventricular conduction.

57- Signs of cardiac tamponade include all of the following except

- a) Paradoxical pulse
- b) Hypotension
- c) Pericardial knock
- d) Tachycardia

Pericardial knock occurs in constrictive pericarditis. It is High pitched early diastolic sound due to sudden halting of the relaxing ventricles by rigid pericardium.

58- Reversed splitting second heart sound occurs in all of the following except

- a) LBBB
- b) AS
- c) ASD
- d) Systemic hypertension

59- Which of the following is an obligatory sign of malignant hypertension

- a) Renal failure
- b) Papilledema
- c) Heart failure
- d) Arterio-venous crossing changes

- Accelerated hypertension : Accelerated → grade 3 retinopathy

- Malignant hypertension : hypertensive crisis with grade 4 retinopathy (papilledema)

60- A hypertensive patient showing a hypertensive crisis in response to propranolol most probably has :

- a) Pheochromocytoma
- b) Conn's syndrome
- c) Renovascular hypertension
- d) None of the above

61- All of the following are used in the treatment of severe left ventricular failure except :

- a) Disopyramid
- b) Nitroprusside
- c) Dobutamine
- d) Mechanical ventilation

Disopyramide is class 1a antiarrhythmic drug & has a negative inotropic effect. Antiarrhythmic drugs are generally -ve inotropic and should be avoided as possible.

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